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ENDEMIC MENINGOCOCCUS MENINGITIS

THE CLINICAL MANIFESTATIONS IN INFANCY AND IN EARLY CHILDHOOD *

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The introduction of an efficacious serum therapy in meningococcus meningitis has made its early diagnosis of supreme importance, particularly in infancy, the age of highest mortality in untreated patients. Early recognition of this type of meningitis is more profitable than in any of the acute infectious diseases, with the probable exception of diphtheria. Experience at the Babies' Hospital demonstrates that this disease had not been recognized in the majority of 136 cases previous to admission to the hospital, although in 134 of these cases the clinical indications were sufficiently apparent to warrant immediate lumbar puncture and diagnosis. We report the clinical manifestations in 136 endemic cases in infants and young children for the purpose of directing attention to the multiform symptomatology of meningococcus meningitis and the necessity of more frequent early examinations of the spinal fluid in those suspected of having the disease.

The variability of the clinical pictures in the epidemic form of meningitis has been emphasized by both early and recent observers. In his monograph published in 1867, Stillé [1] stated that "no disease whatever wears such various masks of symptoms." He referred to this "chameleon-like disorder," [2] and remarked that "no single typical case nor any special description can accurately represent this protean affliction as a whole nor carry a just idea of its numerous grades or multiform aspects." [3] Recently, Herrick,[4] in his discussion of cerebrospinal fever, remarked that "in no acute disease is the course more variable."

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1. Stillé, A.: Epidemic Meningitis, Philadelphia, Lindsay & Blakiston, 1867, p. 17.

2. Stillé (footnote 1, p. 19).

3. Stillé (footnote 1, p. 22).

4. Herrick, W. W.: Cerebrospinal Fever, in Cecil's Textbook of Medicine, Philadelphia, W. B. Saunders Company, 1927, p. 117.

Rolleston and Andrewes [5] commented on the lack of any pathognomonic sign or symptom in this disease. The difficulties of diagnosis are multiplied in nonepidemic periods, particularly in the sporadic cases in early life, chiefly because the physician does not suspect the disease and the age of the patient prevents the expression of his complaints with accuracy.

The following data were obtained from 136 patients with this disease admitted to the Babies' Hospital from 1914 to 1926, during which period the disease had not appeared epidemically in New York City. Accurate medical observation on the symptomatology is possible only during an epidemic, when physicians suspect the disease in its early stages. In endemic cases, we must necessarily rely on the description furnished by parents, which is probably often inadequate. A necessary part of our data is based on such observations, though we realize the probable incompleteness of such a report. However, as such descriptions must be the usual basis for diagnosis, so far as history is concerned, in endemic cases in patients appearing for treatment in the clinic or the hospital, the practical value of their appreciation is manifest.

PREHOSPITAL EXPERIENCE AND MANAGEMENT

That meningococcus meningitis is frequently not recognized is demonstrated by the prehospital experience in our cases. On admission, 134 cases were diagnosed by lumbar puncture and the finding of purulent cerebrospinal fluid with meningococci. Two patients were under observation in the hospital for five and thirteen days, respectively, before the clinical picture seemed to indicate the necessity for examination of the spinal fluid.

CASE 1.—The patient was in the hospital, with the diagnosis of pneumonia, for five days before examination of the spinal fluid revealed a purulent fluid with meningococci.

CASE 2.—The patient was under observation in the hospital for thirteen days before a lumbar puncture was done and a purulent fluid with meningococci found. Previous to lumbar puncture the condition was diagnosed as infection of the upper respiratory tract.

CASE 3.—The patient was examined frequently in the outpatient department for twelve days before admission to the hospital was advised. Fever had been present for eight days; on the eighth day myringotomy was performed, and daily examinations were made thereafter until admission to the hospital on the twelfth day. Lumbar puncture yielded purulent spinal fluid containing meningococci.

In 95 patients, the prehospital management was described in detail; 23 of these had not been seen by a physician previous to admission; 72 had been under medical supervision. Of the 72, 42, or 57 per cent, had

5. Rolleston, H., and Andrewes, F. W.: Cerebrospinal Fever, in Nelson's Loose-Leaf Living Medicine, vol. 2, p. 59.

been observed by the physician for more than three days and the disease had not been suspected; in 31 cases (43 per cent) admission to the hospital for investigation was advised within seventy-two hours of the onset, although meningitis had not been suspected in a considerable number of these. It is apparent from these figures that the diagnosis of endemic meningitis in infancy is too frequently overlooked (in considerably more than 50 per cent of the cases). Extracts from the histories of some of the unrecognized cases are instructive.

CASE 4.—The mother noticed a lump on the child's head (bulging fontanel) fourteen days before admission. The physician told her it was due to teething. Lumbar puncture was not performed.

CASE 5.—The patient was seen by the physician every day for three weeks. The parents were told that the infant "had a touch of meningitis." Lumbar puncture was not performed.

CASE 6.—The patient had a stiff neck for five weeks. He was treated by the physician for vomiting and stomach trouble. Lumbar puncture was not performed.

CASE 7.—Eleven days before admission, the mother noticed that the infant's neck was stiff and the head drawn back. She took the infant to a physician who told her that "stiff neck did not amount to anything." Lumbar puncture was not performed.

CASE 8.—The infant had fever for four weeks. Five days before admission, the mother noticed retraction of the head and took the infant to a physician who told her that the child was "threatened with meningitis." Lumbar puncture was not performed.

CASE 9.—The child had had fever and had vomited for three weeks. On the second day of the disease, it had had a long series of convulsions, and at the same time, retraction of the head and a fine bluish red rash on the skin of the buttocks and extremities were noticed. The parents asked the physician about the retraction of the head and the rash. He diagnosed the condition as food poisoning and mentioned the possibility of meningitis but did not think that the infant had it. Lumbar puncture was not performed. The condition progressed to the chronic basilar type of the disease.

CASE 10.—The child had convulsions and a stiff neck for one month. It was seen daily by the physician who stated that the infant "had a touch of meningitis." Lumbar puncture was not performed.

In these cases, the indications for examination of the spinal fluid seem certain. It is apparent that in some there was a failure to recognize the significance of such signs as a bulging fontanel or stiffness of the neck in meningitis, and it is even more unfortunate that in the cases in which the disease was suspected ("a touch of meningitis" and "threatened with meningitis") the diagnosis was not confirmed by examination of the spinal fluid. Frequently, lumbar punctures are done at the Babies' Hospital to exclude the possibility of meningitis; in spite of this, some patients may have recovered spontaneously without recognition while in the hospital. In two additional cases, lumbar punctures were done, and meningococcus meningitis was not then recognized as the parents were

told that the patient had a fatal disease and serum treatment was not advised or given. One of these patients recovered under serum treatment while in the hospital. We concluded that an erroneous diagnosis of tuberculous or streptococcus or pneumococcus meningitis had been made. The last two cases demonstrate the advisability of serum treatment in any case of meningitis in which the bacteriologic diagnosis is doubtful.

STAGE OF DISEASE AT ADMISSION TO HOSPITAL

Of the 136 patients, 41, or 30.1 per cent, were admitted within seventy-two hours of the onset of known symptoms; 68, or 50 per cent, within the first week of the onset of the disease; 15, or 11.1 per cent, in the second week; 33, or 24.2 per cent, in the third and fourth weeks, while 20, or 14.7 per cent, were not admitted to the hospital until their symptoms had been present more than four weeks.

TABLE 1.—*Age Incidence in One Hundred and Thirty-Six Cases*

Age in Months	Number of Cases	Percentage Incidence
Not over 3 months	24	17.6
3 to 6 months	29	21.3
6 to 9 months	28	20.5
9 to 12 months	11	8.0
1 to 2 years	24	17.6
2 to 5 years	18	13.2
Over 5 years	2	1.4
Total cases	136	

Of 24 infants up to 3 months of age, 12, or 50 per cent, were admitted in the first week of the disease.

Of 29, aged from 3 to 6 months, 13, or 44.8 per cent, were admitted in the first week of the disease.

Of 28, aged from 6 to 9 months, 12, or 42.8 per cent, were admitted in the first week of the disease.

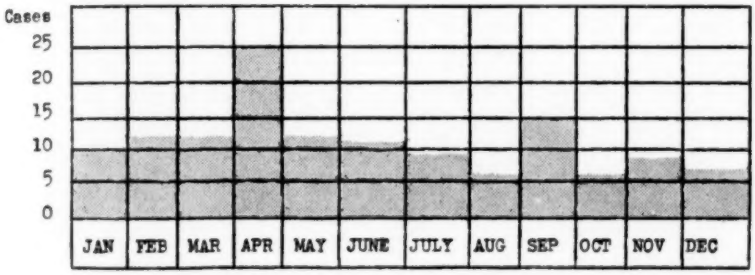
Of 11, aged from 9 to 12 months, 3, or 27.3 per cent, were admitted in the first week of the disease.

Of 44, over 12 months of age, 28, or 63. per cent, were admitted in the first week of the disease.

AGE INCIDENCE

The patients described in this study were all observed at the Babies' Hospital, an institution which admits only infants and young children. On account of the name, it is associated in the minds of the laity particularly with disorders of infants. This may have some bearing on our figures for age incidence. The youngest patient was 23 days old Whether the disease is endemic or epidemic, it is essentially a disease and the oldest, 7 years. Table 1 presents the information in detail.

of early life. Oppenheim [6] mentioned the affinity of the meningococcus for childhood and stated that in one epidemic 82 per cent of the cases occurred in children under 10 years of age and in another, 79 per cent. He noted that persons over 30 seldom have the disease. Westenhoffer [7] affirmed that meningitis is a disease of childhood. Kirschner [8] stated that among 2,037 cases in the Prussian epidemic of 1904-1905, 79.6 per cent of the patients were under 10 years and 48.5 per cent were less than 5 years of age. Flatten [9] stated that of 2,916 cases in the epidemic at Oppeln, 47.4 per cent of the patients were not more than 5 years of age. In the experience of Higier [10] with epidemic meningitis, more cases occur in the first five years of life than in all other ages combined. When these figures relating to the disease when it prevails epidemically are compared with the following, which shows the age distribution in the endemic disease, it appears that young subjects are specially prone to meningococcus meningitis whether the disease is endemic or epidemic, and that the age ratio for the endemic and epidemic types is about the



Seasonal incidence.

same. For a five year period in New York City,[11] from 1921 to 1925 inclusive, 906 cases of this disease were reported, of which 478, or 52.7 per cent, occurred in children under 5 years of age. In the State of New York,[12] outside the city in a four and three-quarter year period, from 1922 to the end of the third quarter of 1926, 402 cases of the disease were reported, of which 162, or 40.2 per cent, were in children under 5 years of age.

6. Oppenheim, H.: Lehrbuch der Nervenkrankheiten **2**:1167, 1923.

7. Westenhoffer: Pathologische Anatomie und Infektionsweg bei der Genickstarre, Berl. klin. Wchnschr. **42**:738, 1905.

8. Kirschner, M.: Ueber die gegenwartige Epidemie der Genickstarre und ihre Bekämpfung, Berl. klin. Wchnschr. **42**:710, 1905.

9. Flatten, H.: Die überstragbare Genickstarre im Regierungsbezirk Oppeln im Jahre 1905 und ihre Bekämpfung, Klin. Jahrb. **15**:211, 1906. Ref. p. 256.

10. Higier, H.: Zur Symptomatologie und Prognose der selteneren Formen epidemischer cerebrospinaler Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **35**:318, 1916-1917.

11. Personal communication from the New York City Department of Health.

12. Personal communication from the New York State Department of Health.

SEX AND SEASONAL INCIDENCE

Seventy-five, or 55.1 per cent, of the cases occurred in boys and 61, or 44.9 per cent in girls. This shows a slightly increased susceptibility in the male sex, as noted by other observers, but the limited number of cases lessens the significance of these figures.

The seasonal incidence is shown in the accompanying figure. The figure depicts graphically the seasonal incidence by months of 136 cases of sporadic meningococcus meningitis. It shows that the disease may occur in any month of the year, but that it has a special predilection for April and some predilection for September. The rise in the spring is probably related to the increased prevalence of respiratory disease at that time, thus making the meningococcus carrier with a superimposed infection of the upper respiratory tract a particularly potent carrier of infection. It might also be explained by the lowering of immunity in infants and young children from infections of the upper respiratory tract, thus making them less resistant to invasion by the meningococcus. The September rise, in all likelihood, is indirectly related to the opening of the schools.

SOCIAL STATUS

Of our entire group of patients, all but three were admitted from neighborhoods of the greatest crowding, poverty and poor hygiene. With two exceptions, the patients that we saw before admission to the hospital were from the slums. This is in contrast to the incidence of this disease during epidemics in which the social status is not so great a factor in immunity.

MATERNAL NURSING

The following information shows that maternal nursing does not favorably influence immunity to meningococcus meningitis. Of 22 infants not more than 3 months of age, 18 were nursed exclusively up to the time of the onset of the disease. Of 27 infants, aged from 3 to 6 months, 21 had been nursed exclusively. Of 28 infants, aged from 6 to 9 months, 15 were still nursing when the symptoms of the disease were first noted.

NUTRITION AS A FACTOR IN IMMUNITY

Since patients who are critically ill are usually not weighed on admission to the hospital, the weights of only fifty-three patients are available for the purpose of learning whether the state of their nutrition was one of the factors which might have had a bearing on lack of immunity It will be noted that many of these children were substandard as to weight. In order to have this considered as a factor in susceptibility, all or nearly all would have to be in the substandard group, since the milieu from which patients with this disease are recruited also furnishes the largest percentage of badly nourished infants and children.

Of the thirty-three children weighed within ninety-six hours of the onset of the disease, twenty were less than 1 year of age; of these, sixteen, or 80 per cent, were more than 10 per cent underweight. Of the thirteen children in this group who were more than 1 year of age, seven, or 55 per cent, were more than 15 per cent underweight.

Of the twenty children weighed within seventy-two hours of the onset of the disease, thirteen were less than 1 year of age; of these, eleven, or 87 per cent, were 10 per cent or more underweight. Of the seven children in this group who were more than 1 year of age, three, or 42 per cent, were 15 per cent or more underweight.

CLINICAL MANIFESTATIONS

Changes in the Sensorium.—Irritability of the sensorium, expressed as fretfulness, a change in disposition and an overresponse to external stimuli, is a significant symptom, but is certainly not pathognomic of the disease, since it may be due to a variety of causes. It is most suggestive when the patient cries out if it is approached, or screams if it is touched. The same response may be elicited in the infant with the skeletal lesion of scurvy, in the widespread chondro-epiphysitis of syphilis, or in acute osteomyelitis. In these, however, a pseudoparalysis of pain is generally present. Göppert [13] stated that "when other signs fail, a cue to the diagnosis is hyperesthesia of the lower extremities, particularly the tenderness on passive motion."

In 44, or 32.8 per cent of our 136 patients, the family had observed that the patient who had previously been normal had, without discoverable cause, become fretful. In 65, or 47.7 per cent, similar manifestations were noted on admission to the hospital. In 84, or 62.5 per cent, of the patients, irritability was noted either by the parents before admission or on the first physical examination. Irritability is one of the most valuable signs to focus attention on the central nervous system. It often alternates with periods of somnolence or stupor. Children may also squirm about in bed, seemingly seeking the position of least discomfort. The type of irritability which to us has seemed most suspicious is that which is produced by any manipulation of the patient and may occasionally even be excited by a near approach to the patient's bed. These patients are hostile to examination, as Debré [14] expressed it, but when left undisturbed they tend to become quiet. He also commented on the extreme degree of "agitation which may be present—incessant crying which may continue day and night—marked cutaneous hyperesthesia—the least

13. Göppert, F.: Meningitis cerebrospinalis epidemica, Handbuch der inneren Medizin **1**:366, 1925.

14. Debré, R.: Recherches épidémiologiques, cliniques et thérapeutiques sur la méningite cérébro-spinale, Paris, Alcan, 1911, p. 50.

touching producing crying." The hyperesthesia of the skin and muscles is often so marked that light touches elicit vigorous reflexes. Even light and sound afford disturbance to some patients.

In our experience, three different types of irritability are encountered before initial lumbar puncture. In the first, the patient is irritable at all times without regard to handling. This is illustrated by the following case:

CASE 11.—An infant, aged 2½ months, had had fever and had been fretful for six days. The mother noted nothing except fever and poor appetite and that the child "cried most of the time."

In the second type, the patient is most irritable when disturbed for examination, attempted feeding, nursing, etc. This is the commonest of all types and is well exemplified by the following case:

CASE 12.—An infant, aged 6 months, had been restless at night and irritable all day for two and one-half weeks. It cried more when picked up or touched. It was referred to the hospital with a diagnosis of colic.

The third type is stuporous and difficult to arouse, but when aroused manifests irritability, as do the other types by crying and screaming, resisting examination. The following case is typical.

CASE 13.—An infant, aged 22 months, had had fever for six days, during which time he slept and objected to being aroused. He did not have convulsions, nor did he vomit or present other symptoms suggestive of involvement of the meninges.

All types of irritability usually show marked relief after lumbar puncture.

Depression of Sensorium.—Forty, or 29.4 per cent, of the patients were drowsy, stuporous or comatose before admission to the hospital. In 29, or 21.3 per cent, similar changes in the sensorium were observed on admission to the hospital. In 63, or 46.2 per cent of 136 patients, drowsiness, stupor or coma was present before admission or was noted at the first physical examination in the hospital. Of 41 patients admitted within seventy-two hours of the onset of known symptoms, 24, or 58.5 per cent, had changes in the sensorium. There is nothing characteristic about the stupor in meningococcus meningitis except that when the patient is aroused, he usually cries from pain and continues to cry until he again becomes stuporous. When the patient is aroused from a stupor resulting from other causes in early life, the change from the stuporous state to that of complete consciousness is usually not painful, and after a variable interval the patient peacefully lapses into his former state. The former type of stupor is illustrated by a patient seen at home by one of us recently.

CASE 14.—A boy, aged 13 months, had been well until three days before admission to the hospital. Without previous warning, a convulsion occurred; at this time, fever was noted. The following day he was stuporous, but awoke crying from time to time when food was offered, which he promptly vomited. When seen late in the afternoon of the third day, he was lying in a stupor with retracted head. The eight members of the family were engaged in many forms of activity in the same room with the patient who was unconscious of it all until disturbed for examination. He cried vigorously throughout the examination and stoutly resisted inspection of his mouth and ears; yet when undisturbed, he immediately relapsed into stupor.

A similar type is the following:

CASE 15.—An infant, aged 9 months, became drowsy twenty-four hours before admission to the hospital; the drowsiness increased; the child could be aroused but when undisturbed was drowsy. The neck was stiff and tender; the child was not irritable.

Muscular Rigidity.—At the time of admission to the hospital, muscular rigidity was detected in 96, or 70.5 per cent of these patients. The parents had noted stiffness of the neck, spasticity of the spine and rigidity of the extremities in 52, or 39.5 per cent, of the patients, in some of whom muscular rigidity was not found at the time of our first examination. Analysis shows that by combining these two groups there were only 21 cases, or 15.5 per cent, in which muscular rigidity had not been manifest at some time previous to lumbar puncture. In 115 cases, or 84.5 per cent, muscular rigidity was apparent either in the history or in the physical examination. This incidence might be further increased by eliminating the 15 fulminating cases in which muscular rigidity rarely occurred.

It seemed possible that this symptom may have developed late in the disease, and that its absence early in the disease might have been responsible for the frequent failures in diagnoses previous to investigation in the hospital. However, in our patients who were seen early (41 within seventy-two hours of the onset), muscular rigidity was present in 30, or 73.1 per cent. Thus, in our experience, this symptom is present in a large majority of cases early and late in the disease.

The most common and significant type of muscular rigidity is spasm of the extensors of the neck which gives rise to stiffness of the neck and retraction of the head. The severity of this symptom varied greatly from extreme opisthotonos to mild involvement, which is easily overlooked. Debré [15] pointed out that meningitis causes a permanent rigidity of the neck, whereas in other conditions one sees intervals of relaxation. This has been true in our patients during the few hours that we have observed them previous to the injection of serum. We believe, however, that muscular rigidity may completely subside after making its appear-

15. Debré, R. (footnote 14, p. 48).

ance. We have seen several instances in which stiffness of the neck and retraction of the head were sufficiently obvious to have been observed by the parents some days before examination and in which muscular rigidity could not be detected when we first saw the patient.

Infants and young children have more flexible spines than adults. For this reason, it is easier to detect rigidity in young patients. Tenderness of the neck, on the contrary, is difficult to elicit in infants, as these patients cannot verbally localize the tenderness and as many of them with general hyperesthesia cry out if manipulation of any part of the body is attempted. Both Debré [15] and Worster-Drought and Kennedy [16] emphasized the importance of careful examination for this symptom. The latter authors suggest the following technic:

> With the patient lying on his back, the observer places the hand beneath his head and endeavors to gently draw it forward—in the presence of meningitis, except occasionally in the tubercular form, the head cannot be brought forward more than two inches beyond the vertical line of the long axis of the body. With manipulation of the head, true neck rigidity, as met with in meningitis, tends to increase.

When examination is made to determine the rigidity of the neck, the insistence of these patients to lie on their side and their discomfort in being placed in supine position are valuable leads to involvement of the neck. The thighs may be either flexed or extended in varied degrees of spasticity.

The pathogenesis of spasticity of the neck and muscular rigidity in general is not known for a certainty. Foster and Gaskell [17] offered evidence that it is not of local muscular origin because of the relief afforded by lumbar puncture. They noted that "it is greatly diminished and entirely relieved by evacuation of considerable quantities of cerebrospinal fluid." They concluded that increased intracranial pressure is at least a partial factor. Worster-Drought and Kennedy [18] thought the "actual cause of opisthotonos lies probably in the irritation of the posterior spinal nerve roots by inflammatory exudate." Cecil Wall [19] believed that this symptom is due to changes inside the skull in the posterior fossa and said that it may be produced as follows: "In flexion and extension of the head, the cerebellum slides up and down the posterior surface of the medulla, folding and infolding the arachnoid which forms the bridge between them. If the arachnoid be inflamed the pos-

16. Worster-Drought, J., and Kennedy, A. M.: Cerebrospinal Fever, London, Black, 1919, p. 108.

17. Foster, M., and Gaskell, J. F.: Cerebrospinal Fever, Cambridge, University Press, 1916, p. 21.

18. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 110).

19. Wall, Cecil: A Lecture on Cerebro-Spinal Fever, Lancet **1**:1211, 1907.

terior muscles of the neck contract and inhibit such movement." Oppenheim [20] also agreed "that it may be attributed to meningitis of the posterior fossa." Busse [21] explained retraction of the head as a compensatory posture to increase the volume of the subarachnoid space.

Though the exact pathogenesis of muscular rigidity is in doubt, its frequency and importance in meningitis are verified by all observers. We emphasize it as the most important single clinical manifestation of the disease. It should always be carefully searched for in acute disease, and its presence or a history of its occurrence always makes examination of the spinal fluid advisable.

Kernig's Sign.—Kernig's sign, so important in meningitis in adults, is of comparatively little value in early life. This sign was present in 41 per cent of our cases. Age is apparently not a factor in its incidence, since the sign was present in 41 per cent of the patients less than 6 months and in 42 per cent more than 6 months of age. We agree with Worster-Drought and Kennedy that this sign is of little value in children younger than 2 years. We have found it present in a considerable percentage of normal infants and were unable definitely to elicit it in more than half of our patients with meningitis. Its presence, while more important than its absence, leads only to a more careful watch for other signs, and certainly the absence of a positive Kernig's sign should never delay examination of the spinal fluid in a patient suspected of having meningitis. Worster-Drought and Kennedy [22] observed that many explanations have been advanced to account for this sign, but that the most probable is as follows:

> In attempting to extend the leg, traction through the medium of the great sciatic nerve is applied to the spinal nerve roots in the lumbar and sacral regions, and thence is transmitted to the spinal meninges. To inhibit this traction a reflex contraction of the ham string muscles occurs.

Brudzinski's Sign.—Brudzinski's sign was present in 36 per cent of this group of patients with meningitis. We believe that the presence of a sharp definite flexion of the legs at the knees and thighs when the head is flexed on the chest is of real significance as pointing to meningeal disease. However, sharp definite reactions are extremely difficult to obtain in infancy. The eliciting of Brudzinski's sign demands a certain amount of "cooperation" from the patient which is frequently not attainable in infants. Our chief difficulty has been in maintaining extension of the legs when the patient is placed supine before an attempt is made to flex the head. In a considerable percentage of normal and sick infants, there is a tendency to flex the knees and thighs when the patient is placed

20. Oppenheim, H.: Textbook of Nervous Diseases **2**:755, 1911.
21. Busse, O.: Die übertragbare Genickstarre, Klin. Jahrb. **23**:368, 1910.
22. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 116).

supine before any manipulation of the head is attempted. This prevents the carrying out of the test. Also, marked retraction of the head and extreme cervical rigidity effectually prevent flexion at the neck and the sign cannot be elicited.

The definite presence of this sign in patients in whom it can be satisfactorily attempted is highly significant and indicates immediate examination of the spinal fluid. However, it is not possible to try out this sign satisfactorily in a considerable percentage of patients, and in many the flexion response of the legs is so slow as to leave the examiner uncertain as to its presence or absence.

The Reflexes.—The variability of the tendon reflexes in healthy infants greatly detracts from the value of these reflexes in diagnosis. The same variability of tendon reflex response exists in infants with nonmeningeal diseases. Also the changing emotional states in a normal or ill infant will alter the tendon reflexes from time to time in the same person. Normal infants, excited by the manipulation of examiners in unfamiliar surroundings and by strangers, will usually yield hyperactive tendon reflexes. Increased tendon reflexes were present in 59 per cent of these patients with meningitis. Undoubtedly, the tendon reflexes are frequently increased in meningitis but since they are frequently increased in conditions other than meningitis, they have no real diagnostic value in the average sick, irritable infant. However, the presence of moderately exaggerated tendon reflexes in a quiet or somnolent patient, who is acutely ill, is usually of considerable significance and indicates the necessity of examination of the spinal fluid. The cutaneous reflexes, i.e., abdominal, cremasteric and plantar, have not been of assistance in diagnosis.

Bulging of the Fontanel.—In eight instances, or 5.8 per cent, it had been noted by the parents that bulging of the fontanel was present. This was usually termed a "lump on the head." This is mentioned as evidence of the magnitude of the bulging, which gave ocular evidence of its presence. In contrast to the small number of cases in which bulging of the anterior fontanel was noted before admission to the hospital is the relatively large number in which this manifestation was found by either inspection or palpation at the first medical examination, i.e., in fifty-four, or 39.7 per cent, of the patients. The following figures demonstrate the decreasing prevalence of this physical sign after the ninth month coincident with the diminishing size of the fontanel with advancing age.

In 24 infants up to 3 months of age, it was noted in 14, or 58.3 per cent.

In 29, aged from 3 to 6 months, it was noted in 16, or 55.1 per cent.

In 28, aged from 6 to 9 months, it was noted in 18, or 64.2 per cent.

In 11, aged from 9 to 12 months, it was noted in 4, or 36.6 per cent.

In 44, more than 12 months of age, it was noted in 2, or 4.5 per cent.

In 56.6 per cent under 1 year of age, bulging of the fontanel was present.

Bulging of the fontanel is an indication that the ventricles are distended with fluid; hence, an acute hydrocephalus or pyocephalus is present. If the fontanel is sufficiently open, bulging persists until it is relieved by lumbar puncture, spontaneous recovery or death. Bulging of the fontanel is one of the most reliable indications of meningeal infection whether caused by the meningococcus or by any of the pyogenic organisms (we have also seen it in pachymeningitis hemorrhagica), but it does occur in other acute extrameningeal infections of infancy, particularly in pneumonia and acute otitis media.

Infants should be put in the sitting position to determine whether the fontanel bulges, as when they are supine it may be deceiving. Bulging of the fontanel is always an indication for lumbar puncture.

Convulsions.—Convulsions were observed in 40, or 29 per cent, of the cases. They usually occurred several hours or days previous to admission and not infrequently were noted only at the onset of the disease. We have rarely observed the convulsive seizures ourselves. A convulsion that occurred at the onset only is illustrated by the following case, in which the patient was recently seen by one of us.

CASE 16.—In an infant, aged 14 months, the onset was abrupt, with convulsions and hyperpyrexia for eighteen hours. For the following two weeks, the child had fever, the temperature rising at times to 104. Convulsions did not recur. The only suspicious physical symptom on the fourteenth day was slight rigidity of the neck. Lumbar puncture revealed the diagnosis.

After diagnostic lumbar puncture and during treatment, convulsions have occurred in a few of the fatal cases, but in none of the patients who recovered. In our experience, convulsions occur early in the disease, nearly always before aid is sought in the hospital. The convulsions described by parents and seen by us reveal nothing to distinguish meningococcus meningitis from other diseases in which convulsions occur.

If increase in intracranial pressure is the important factor in the causation of convulsions, one would expect the incidence of convulsions to increase as the fontanel becomes smaller and the skull more rigid. Table 2 demonstrates the frequency of convulsions in our group of cases. In those under 1 year of age, 33.5 per cent had convulsions. It can be seen that convulsions are somewhat less frequent after the first year, which indicates that increase in intracranial pressure is only one factor in the etiology of convulsions.

It must be remembered that the instability of the nervous system is greatest at birth, and that it gradually decreases during the first two years of life, as the power of inhibition is established. After the stage of irritation in meningitis has passed and stupor develops, the convulsions no longer occur.

Convulsions may be due to any of the following causes: (1) the toxemia that accompanies the nasopharyngitis and otitis media, which are frequent precursors of the meningeal invasion; (2) early meningococcemia and its accompanying toxicity, and (3) increased intracranial pressure due to inflammatory invasion of the meninges; to metastases to the brain tissue, i.e., encephalitis and, rarely, abscesses of the brain, or to distention of the ventricles with pus or cerebrospinal fluid.

Whether latent or active, tetany may be an etiologic factor in infancy in any of the phases of the infection already described. It may be present with sufficient frequency to have an influence on the data for this symptom.

TABLE 2.—*Frequency of Convulsions in Authors' Group of Cases*

Age in Months	Number of Cases	Number with Convulsions	Percentage
0 to 3	34	10	41.6
3 to 6	29	6	20.6
6 to 9	28	10	35.7
9 to 12	11	5	45.4
12+	44	9	20.4
Total	136	40	29.4

Rolleston [23] and others are the authorities for the statement that the principal lesion of meningococcus meningitis is an exudate at the base of the brain. Göppert [13] found that in the most severe cases, in addition to the basilar exudate, the anterior two thirds of the convexity was covered with a purulent exudate. Since the cerebral cortex, as expressed by Oppenheim,[24] is the chief birthplace of convulsions, it is surprising that they do not occur more frequently. It is interesting to note that among 45 infants from the Babies' Hospital with proved tuberculous meningitis 20, or 44.4 per cent, had convulsions. In a contrasting series of 53 cases of pneumococcus meningitis from autopsy protocols from the same institution, in which the exudate is usually on the convexity rather than the base of the brain, convulsions occurred in 26, or 49 per cent. Table 3 shows the occurrence in the three diseases. When the figures in the table showing the incidence of convulsions are compared with the pathologic conditions in the three diseases, it becomes

23. Rolleston and Andrewes (footnote 5, p. 24).

24. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, 1923, Berlin, S. Karger, vol. 1, p. 81.

evident that the situation of the exudate is only one factor which determines their frequency.

We are inclined to believe that, not infrequently, localizing symptoms may be due to the widespread encephalitis accompanying meningococcus meningitis, recently emphasized by Goldstein [25] and Herrick,[26] as well as to the lesions in the meninges themselves.

The following case, which showed localized convulsions, cannot be explained on the basis of a purulent exudate on the cortex since none was present; it probably cannot be explained on the basis of toxicity, since convulsions of toxic origin in early life are usually generalized, nor was it due to any gross lesion of the motor zone, since the only one found was posterior and behind the accepted motor zone area. It seems, therefore, that in this case microscopic section might possibly have revealed an area of deep encephalitis.

CASE 17.—*History.*—A child, aged 3 years, was admitted to the hospital because of vomiting and drowsiness of twenty-four hours' duration. Stupor alternating with irritability was present on admission. Several hours later, convulsive

TABLE 3.—*Incidence of Convulsions*

	Number of Cases	Percentage of Convulsions
Meningococcus meningitis	136	29.4
Tuberculous meningitis *	45	44.4
Pneumococcus meningitis	53	49.0

* Occurrence prior to admission to hospital.

movements of the left arm and face and nystagmus were observed; these continued for twenty-four hours. Meningococci were found in the blood culture and in the cerebrospinal fluid. Death occurred seventy-two hours after the first symptoms.

Autopsy.—The brain showed a slightly turbid pia-arachnoid without any diffuse inflammatory exudate. Over the motor areas, especially, the membranes were clear. One area, 1 by 1 cm. in diameter, was present over the right first occipital lobe where there was thickening, grayness and possibly mild inflammation. The base was normal, as was the spinal cord in the cervical region. Over the dorsal region and over the cauda equina, the pia-arachnoid was cloudy, and a thin layer of pus was present. The ventricles were normal. The choroid plexus did not show any lesion. The sinuses contained soft clots. Hemorrhages were not present at any point. When the brain was opened and serially sectioned, abscesses or other lesions were not found in any part of its substance.

When considering the etiology of convulsions, one must not forget that the infant is prone to have convulsions from trivial causes and that many of the convulsions in early life are of unknown origin. Infancy as a condition in itself lowers the threshold for convulsions. In infancy

25. Goldstein, K.: Die einzelnen Erkrankunmgen des Gehirns und seine Häute, Handbuch der inneren Medizin, ed. 2, Berlin, Julius Springer, 1925, vol. 15, p. 191.

26. Herrick (footnote 4, p. 115).

and childhood, however, an unexplained convulsion usually indicates a diagnostic lumbar puncture. Unless this is done more frequently, many cases of meningitis in early life will remain undiagnosed.

The Eruption.—Since the term "spotted fever" was first used in the United States to describe epidemic cerebrospinal meningitis, it is interesting to note that in our patients with the endemic form of the disease, 15, or 11 per cent, had a hemorrhagic eruption. In 44 epidemic cases in early life reported by Göppert,[27] 4, or 9 per cent, of the patients had similar eruptions. Although it is generally agreed that hemorrhagic eruptions are more frequently present in older children and adults, three of our patients were not over 6 months of age. Thirty-two and three-tenths per cent of our patients were over 12 months of age, and in this older group of forty-four cases, the percentage was 27.2.

In compilations of cases reported from hospitals, it may be found that there was a low incidence of eruption, because, first, patients with profuse purpura usually represent the severer types of the disease and are therefore more likely to die before treatment in the hospital is sought; second, endemic meningitis is a disease of the slums, in which lives the section of the population most diffident about seeking relief in a hospital in the early stages of disease when hemorrhagic eruptions are most likely to be present. We receive the impression that a number of patients with the specific eruption die before the question of hospitalization is considered, and others have a transitory eruption only in the early stages before admission to the hospital. The following case illustrates the last statement:

CASE 18.—One of us was called to see an infant, aged 9 months, on account of drowsiness for thirty-six hours, vomiting for twenty-four hours and convulsions for twelve hours. About a dozen petechial spots were noted on the legs and ankles. Information was elicited from the parent that at the onset of the disease the "spots on the legs" were much more numerous. Twelve hours later, or forty-eight hours after onset, the eruption had almost entirely disappeared.

It is difficult to compare the incidence of hemorrhagic eruption in the endemic form with that in the epidemic form of the disease on account of the variability of the specific eruption in different epidemics. That this difficulty is not new is shown by the report of the Massachusetts Medical Society of 1809[28] on "Spotted or Petechial Fever" in the New England epidemic, in which it is stated that "it is not easy to determine in how large a proportion of subjects the skin is affected with spots and eruptions."

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27. Göppert, F.: Zur Kenntnis der Meningitis cerebrospinalis epidemica, Klin. Wchnschr. **42**:644, 1905.

28. Report Respecting a Disease Commonly Called Spotted or Petechial Fever, Communications, Mass. M. Soc. **2**:132, 1809.

For further information relating to the skin manifestations of the early epidemics, the essay of Webber [29] should be consulted. Of 164 cases reported by him, 34.6 per cent showed a petechial eruption. Councilman, Mallory and Wright [30] stated that "lesions of the skin seem to have played a greater part in the descriptions of the early epidemics than in the more recent." Neal [31] mentioned that in the epidemic in New York City in 1904-1906 and in her own sporadic cases the incidence of eruption was about 15 per cent.

The 15 cases presenting the petechial or purpuric forms of eruption already mentioned, which occurred in our group of 136 cases, present a clinical manifestation of the first importance. Foster and Gaskell [32] stated that the petechial eruption may be regarded as the true specific rash of the disease, though it is probably the least regular of all the exanthems. A variety of eruptions have been described by numerous authors in reports on cerebrospinal meningitis, but we do not place great importance on their value as leading to diagnosis in infancy and childhood, and we have concerned ourselves only with the hemorrhagic eruption which is the only one characteristic of the disease.

Infants and young children, as compared with adults, are more prone to have transitory eruptions in the presence of fever; in some there may not be any other background than a mild digestive disturbance. If one keeps this in mind, it seems unwise to attach great importance to any but the hemorrhagic eruptions due to bacterial emboli which are a certain indication of a blood stream infection. Meningococci may be demonstrated in the cutaneous lesions by stained sections, as described by Brown.[33] Worster-Drought and Kennedy [34] stated that meningococci may be seen occasionally in blood films taken from the large purpuric areas sometimes called vibices. Netter and Salame [35] demonstrated meningococci in smears of the blood and tissue juices made from purpuric areas. We have recently found meningococci in blood films from a purpuric skin lesion in an infant who recovered. Worster-Drought and

29. Webber, S. G.: Cerebrospinal Meningitis, Boston M. & S. J. **75**:34, 1866.

30. Councilman, W. T.; Mallory, F. B., and Wright, J. H.: Epidemic Cerebrospinal Meningitis and Its Relation to Other Forms of Meningitis, Boston, Mass., State Board of Health, 1898, p. 135.

31. Neal, J. B.: Epidemic Meningitis, in Abt. I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925, vol. 6, p. 422.

32. Foster, M., and Gaskell, F. F. (footnote 17, p. 19).

33. Brown, C. L.: The Skin Lesions in Meningococcus Septicemia, Am. J. Dis. Child. **27**:598 (June) 1924.

34. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 98).

35. Netter, A.: Développement d'un zona dans le domaine du plexus lombaire et du plexus sacré, à l'occasion d'une méningite cérébro-spinale, Compt. rend. Soc. de biol. **79**:755, 1916.

Kennedy [36] reported a case in which meningococci were cultivated from the vesicle forming on the site of a purpuric patch. The large purpuric patches or vibices in these cases most frequently occurred on the back, buttocks and hips. Worster-Drought and Kennedy noted that this type of rash is encountered only in fulminating or in very acute forms and almost invariably foreshadows a fatal result. They mention five patients with fulminating cases with a purpuric eruption who died within forty-eight hours of the onset; in each case, in addition to petechiae, large plum-colored vibices were present on the trunk. In the two following cases, the patients had petechiae, purpura and vibices and recovered after vigorous introduction of antimeningococcus serum both intravenously and intraspinally. In both cases, meningococci were cultivated from the blood and cerebrospinal fluid.

CASE 19.—An infant, aged 18 months, eight days prior to admission to the hospital, had an acute onset of fever, vomiting, drowsiness and diarrhea. These symptoms continued for three days; then for the following three days, restlessness and irritability were present, and on the sixth day "black spots" appeared on the face, buttocks and shoulders. This child presented a typical picture of meningococcus sepsis when seen on the eighth day, with a profuse petechial and purpuric eruption and a metastatic iridochoroiditis, which previously was reported in detail by McLean and Gilmartin.[37]

CASE 20.—In an infant, aged 10 months, diarrhea began four days prior to admission; on the fourth day of the diarrhea, fever, rapid respirations and convulsions occurred. Toward the end of the fourth day, eruption was first noted. On admission to the hospital a few hours later, the patient was cyanotic with rapid respiration. On the face, trunk, buttocks and extremities were numerous petechiae and ecchymotic areas of varying size, one of which was 1½ inches (3.7 cm.) in diameter. From this lesion, meningococci were found in blood films.

The following case illustrates that petechiae may persist in infants who have not been treated.

CASE 21.—An infant, aged 5 months (this was a recent case not included in the group of 136), was admitted to the Babies' Hospital because of fever, irritability and rash of three weeks' duration. Over both legs, thighs, hips, buttocks, arms and forearms were numerous fine petechial spots which closely resembled insect bites. The right elbow joint was swollen and painful on passive motion. Purulent cerebrospinal fluid was obtained by lumbar puncture and was found to contain meningococci. Blood culture was sterile. Antimeningococcus serum was administered both intravenously and intraspinally, and after forty-eight hours the eruption had almost entirely faded, and the swelling of the joint subsided.

It is our impression that the infants with limited crops of petechial spots, of which we have seen several instances, do not represent an especially severe type of the disease. (In fact, patient 21 apparently

36. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 96).

37. McLean, S., and Gilmartin, H. A.: Cerebrospinal Meningitis with Purulent Irodochoroiditis, with Recovery, Am. J. Dis. Child. **27**:603 (June) 1924.

had the petechial spots for three weeks and yet during this long period was not given specific treatment.) This type indicates that the blood stream continued as a reservoir for the growth of the organisms after the meninges were invaded. Such patients do not always show manifestations of toxemia. The period of contamination of the blood stream by the meningococcus is apparently variable in duration, transitory in some cases and of long duration in others. It may occur at the onset only. It may be noted after the onset of meningeal symptoms. It may be the first clinical manifestation and may continue throughout the course of the meningeal invasion. Examples of all three types are included in our series of cases.

Herpes.—Herpes was observed in only two of our entire group of patients; both patients were over 12 months of age. Göppert [38] stated that he had not seen it in patients under 3 years of age in either endemic or epidemic meningitis. In the epidemic in upper Silesia, Göppert [39] reported that among forty cases in his private practice three children, aged 5½, 7 and 8 years, had herpes. In the same epidemic, Higier [40] noted herpes frequently in old persons; he seldom saw it in children and never in infants. Debré,[41] who did not state how frequently he had observed herpes in young subjects, noted that it is less frequent in infancy than in early life. Crothers [42] stated that herpes is common. Holt and Howland [43] noted that herpes is present on the lips and face commonly in older children, but that it is rare in infants. Herford [44] found meningococci in pus taken from herpetic lesions. From our own experience and that of others who have had ample opportunity to see large groups of patients, it may be stated that in both the endemic and epidemic forms of the disease herpes is rare in infancy, but may occur in early childhood. Its presence is a diagnostic aid; its absence is without significance.

Fever.—In our experience, there is no temperature curve which in any sense is characteristic of the disease. The febrile reaction is seldom a guide to the severity of the disease. Severe forms, particularly in

38. Göppert, F. (footnote 13, p. 358).

39. Göppert, F.: Zur Kenntnis der Meningitis cerebrospinalis epidemica, Berl. klin. Wchnschr. **42**:646, 1905.

40. Higier, H.: Zur Symptomatologie und Prognose der selteneren Formen epidemischer cerebrospinaler Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **35**:320, 1916-1917.

41. Debré, R. (footnote 14, p. 52).

42. Crothers, B.: Disorders of the Nervous System in Children, New York, D. Appleton & Co., 1926, p. 89.

43. Holt, L. E., and Howland, J.: Diseases of Infancy and Childhood, ed. 9, New York, D. Appleton & Co., 1926, p. 597.

44. Herford: Bakteriologische und epidemiologische Beobachtungen bei einer Genickstarreepidemie in Altona, Klin. Jahrb. **19**:265, 1908.

infancy, may occur with little fever, and young subjects may go through the entire course of the disease with only slight febrile reaction.

In 102, or 75 per cent, of our cases fever had been noted by the parents prior to observation in the hospital. In some, it doubtless escaped observation. Of 131 patients, records of whose temperature were available during the first forty-eight hours of observation, 29 were afebrile during this period; 28 of these yielded purulent cerebrospinal fluid at initial lumbar puncture. Of these 29, 3 were admitted in the first seventy-two hours of the disease, 3 from the fourth to the seventh day of the disease, and the remaining 23 after symptoms had been present for at least one week. Many of the last mentioned had had symptoms for several weeks; 15 of this group gave a definite history of fever in the earliest stages of the disease.

Although fever is usually present at some stage of the disease, occasionally the patient may have little elevation of temperature throughout the course of the infection, as exemplified by the two following cases.

CASE 22.—A girl, aged 4½ years, was admitted on the third day of the disease. The temperature had been taken several times at home, and at no time had fever been present. For the first sixty hours after admission, she was afebrile.

CASE 23.—An infant, aged 4 months, without a history of fever, was admitted on the twelfth day of the disease; the cerebrospinal fluid was purulent. The patient was afebrile during the first seven days in the hospital. The patient did not improve when specific serum therapy was employed.

Age does not have a striking influence on the febrile reaction, as the following figures demonstrate: In 53 patients not more than 6 months of age the parents noted fever in 69.8 per cent, while in 83 patients more than 6 months of age fever was thought to have been present in 78.3 per cent.

Debré [45] observed that fever is irregular and that the temperature varies from one moment to another. Herrick [46] and Griffith [47] confirmed this statement. Debré noted the difference of a "degree or two" in temperature measured at short intervals. We agree with him that a high temperature is rare in cases which terminate favorably; we have also had cases terminate unfavorably in which fever had not been present while the patient was under observation. Debré [48] described "hypothermia" as occurring in some cases, especially in the first day after which the fever rose to the usual elevation. We have recently seen a patient, aged 14 months, with 740 cells and viable organisms who was afebrile. Absence of fever should not be used as an argument against diagnostic lumbar puncture.

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45. Debré, R. (footnote 14, p. 68).

46. Herrick, W. W.: Meningococcus Infections, in Osler and McCrae: Modern Medicine **1**:576, 1925; footnote 4, p. 116.

47. Griffith, J. P. C.: Diseases of Infants and Children **1**:422, 1919.

48. Debré, R. (footnote 14, p. 53).

GASTRO-INTESTINAL SYMPTOMS

Vomiting.—Of the gastro-intestinal disturbances occurring in meningococcus meningitis in early life, vomiting is the only symptom which has diagnostic significance.

In our experience, the vomiting in meningococcus meningitis in infancy and early childhood is identical with that observed in other acute infections and in gastro-intestinal disorders due to improper feeding. We have not observed the so-called cerebral vomiting sometimes described as occurring in meningococcus meningitis. We have not been able to differentiate the vomiting in our patients from that due to a wide variety of conditions in early life, and it has not had a projectile character in any instance. The following case typifies a number of our histories.

CASE 23.—A breast-fed infant, aged 6 months, was brought to the office of one of us because of vomiting which had occurred after nursing for the preceding seven days. A physician had been treating the infant for "gastritis." There was nothing noteworthy about the vomiting. Sleep was frequently disturbed. Meningococci were found in the cerebrospinal fluid.

TABLE 4.—*Occurrence of Vomiting* *

Age in Months	Total Number of Patients	Number that Vomited	Percentage that Vomited
0 to 3	24	9	35.5
3 to 6	29	16	55.1
6 to 9	28	16	57.1
9 to 12	11	9	81.8
12+	44	27	60.9

* Vomiting occurred in 77 of 136 cases, or in 56.6 per cent.

The character of the vomiting in infancy is too seldom a guide to its cause, and one should never wait for projectile vomiting to indicate the necessity for diagnostic lumbar puncture. As in other clinical manifestations of meningitis, the severity of this symptom varies greatly from a single emesis at the onset to repeated vomiting throughout the course of the disease, generally immediately after the ingestion of food.

Since vomiting occurred in only 56.6 per cent of our cases, as shown in table 4, its absence should not be a deterrent to diagnostic lumbar puncture.

Diarrhea.—In the French literature are numerous references (Debré [49]) to diarrhea as a symptom of the early stages of meningococcus meningitis in certain cases. In only three of our cases, however, was this symptom mentioned by the parents as one of the reasons for seeking relief at the hospital. It is possible that the actual incidence of diarrhea was higher than 2 per cent, since the more unusual symptoms,

49. Debré, R. (footnote 14, p. 65).

such as drowsiness, etc., would naturally be stressed. It is also possible that those who stress the importance of diarrhea as a symptom have been overcredulous, since at the first appearance of illness the infant is usually given a purge and therefore the medication rather than the disease may have been the cause of the diarrhea. Many parenteral infections begin with diarrhea, but in our experience neither more nor less frequently than those of the meninges.

In nine of our patients, loss of appetite was one of the reasons for seeking observation in the hospital.

In describing the epidemic disease, a number of observers mentioned the intestinal lesions in fulminating cases; others observed the presence of bloody stools in severe and fulminating cases. Denehy [50] stated that in one of his cases a blood clot filled the intestinal canal, but that there was no lesion in the intestinal wall to explain the hemorrhage. Horder [51] noted that small hemorrhages in the intestinal canal may be conspicuous, especially in acute cases. Wilson [52] found hemorrhages into the bowel wall and swelling of the lymphoid tissue of the intestine. MacLagan and Cooke [53] also discovered petechial hemorrhages into the intestinal mucous membrane. In a large series of autopsies performed on children who had had the epidemic form of the disease, Göppert [54] found in a number of fulminating cases punctuate hemorrhages in the stomach, and in the large and small intestine, swelling of Peyer's patches and solitary follicles and inflamed areas in both the large and the small intestine.

The two following cases (the only ones in our series in which bloody stools were present) demonstrate that bloody stools may occur in other types than the fulminating, since both of these patients recovered. In neither case was the melena a diagnostic aid, since in both cases the diagnosis was apparent. It seems probable that hemorrhages into the bowel are to be found only in cases presenting more classic signs of the disease. They are of such rare occurrence (1.4 per cent of our cases) that their presence might tend to confuse rather than aid in the diagnosis of the disease.

CASE 24.—An infant, aged 22 months, was brought to the hospital on account of vomiting, petechial eruption, diarrhea with tarry stools, drowsiness of two days' duration, strabismus and retraction of the head of less than one day's duration. In addition to the petechial eruption, opisthotonos, bilateral internal

50. Denehy, W. J.: Epidemic Cerebro-Spinal Meningitis, Brit. M. J. **2**:689, 1916.

51. Horder, T. J.: Cerebro-Spinal Fever, London, Frowde, 1915.

52. Wilson, W. J.: Certain Points Observed with Regard to Cerebro-Spinal Fever in the Belfast Military District, Brit. M. J. **2**:904, 1916.

53. MacLagan, P. W., and Cooke, W. E.: The Fulminating Type of Cerebro-Spinal Fever, Brit. M. J. **2**:869, 1916.

54. Göppert, F.: Zur Kenntnis der Meningitis cerebrospinalis epidemica mit besonderer Berücksichtigung des Kindesalters, Klin. Jahrb. **15**:533, 1906.

strabismus, changes in sensorium, etc., were observed at the first examination at the hospital. Meningococci were present in the smears and cultures from the purulent cerebrospinal fluid.

CASE 25.—An infant, aged 15 months, was brought to the hospital because of vomiting and fever of three days' duration, purpuric eruption of two and one-half days' duration and tarry stools, one of which was passed during examination. A profuse purpuric eruption, apathy, rigidity of the neck, retinal hemorrhage, etc., were present on admission to the hospital. Meningococci were found in cultures from both the blood and the cerebrospinal fluid.

INSTABILITY OF THE VASOMOTOR SYSTEM

Instability of the vasomotor system as manifested by spontaneous flushing in different parts of the skin, evanescent localized erythema, and tache cérébrale is frequently present in all the types of meningitis. Vasomotor instability is present in so many acute infections of infancy and childhood that there seems to be little justification in its present association with meningitis. Tache cérébrale is recorded in meningitis more frequently than in other types of acute infection, perhaps because the search for this sign is more regularly made in meningeal than in extrameningeal disease. Tache cérébrale was present in 42, or 30.8 per cent of our cases, but it was present in 31, or 51.6 per cent of 60 infants and young children hospitalized on account of extrameningeal disease.

Worster-Drought and Kennedy [55] observed that disturbance of the vasomotor system is noted in all cases. They found tache cérébrale invariably well marked in their cases, but they believed that it does not have diagnostic value since it is by no means confined to cerebrospinal fever. We have occasionally seen it months and years after recovery from the disease.[56] They noted that it was present months after convalescence. In addition to the signs of vasomotor instability before noted, they also observed irregularities of perspiration.

Netter and Debré [57] suggested that vasomotor instability is due to irritation of the floor of the fourth ventricle, the ependymal lining of which frequently shows marked changes. Worster-Drought and Kennedy [55] believed that it is more likely a result of asthenia, as a tache cérébrale can be elicited in most cases of neurasthenia and other conditions in which the nervous system is depressed.

INVOLVEMENT OF THE EYES

Involvement of the eye occurred with sufficient frequency to warrant comment, as table 5 indicates.

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55. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 129-130).

56. McLean, S., and Caffey, J. P.: Sporadic Meningococcus Meningitis; Sequelae Following Specific Serum Therapy in Infancy and Early Childhood, J. A. M. A. **87**:91 (July 10) 1926.

57. Netter, A., and Debré, R.: La méningite cérébro-spinale, Paris, Masson et Cie, 1911, p. 242.

Of these symptoms, strabismus is the most important. It was mentioned thirteen times, or in 9.5 per cent of 136 cases, as one of the symptoms which urged the parents to seek relief at the hospital. As mentioned before, although lay observations are open to suspicion, the history of a sudden onset of ocular palsy not previously observed cannot be taken lightly even in infancy. On the first physical examination in the hospital, it was present in 23, or 16.9 per cent, of the patients. If the number of those with strabismus on admission is added to the number of those with a history of strabismus which was not present on the first physical examination, it will be seen that 29, or 20.1 per cent, had strabismus before admission to the hospital. Age would not seem to have any bearing on the incidence, since in infants under 6 months of age it was observed in 15 per cent; in those from 6 to 12 months, 25.6 per cent, and in those over 1 year, 11.3 per cent.

Strabismus is more frequently associated with the symptomatology of tuberculous than with meningococcus meningitis. That the difference

TABLE 5.—*Incidence of Disturbances of the Eye*

	Occurrence	Percentage
Strabismus	29	20.1
Nystagmus	14	9.7
Pupillary changes	12	8.3
Conjunctivitis	2	...
Iridochoroiditis	3	2.2
Photophobia	1	...
Optic neuritis	1	...
Retinal hemorrhage	1	...
Stare	6	4.4

in the incidence is not sufficiently striking to be of great value in the differential diagnosis of the two diseases is demonstrated in the following statement: Of 44 patients with tuberculous meningitis from the Babies' Hospital in whom the diagnosis was verified by autopsy or by finding tubercle bacilli in the spinal fluid, 13, or 29.5 per cent, had strabismus. Worster-Drought and Kennedy [58] stated that "disturbances of the extrinsic muscles of the eye are often observed in cerebrospinal fever, but not so constantly as in tuberculous meningitis. Strabismus, when present, is usually divergent and if paresis occurs, the sixth nerve, no doubt owing to its long intracranial course, is the nerve most usually involved."

Nystagmus was present in 11, or 8 per cent, of our cases on the first physical examination. If we add this number to the cases in which it was noted only prior to admission, it is increased to 14, with a percentage of 9.7 in 136 cases. This is a sign of little value in diagnosis, first, because of its infrequency; second, because it occurs in many extra-

58. Worster-Drought, J., and Kennedy, A. M. (footnote 16, p. 62).

meningeal diseases; and third, because a variety of disorders of the central nervous system involving different areas may cause it.

Pupillary changes—unequal, pinpoint, fixed, sluggish—were present in 12, or 8.3 per cent of the patients. The possibility of this condition being caused by the administration of drugs before admission is not excluded. Three patients had metastases in the eye manifested as iridochoroiditis. We regret that our ophthalmoscopic examinations were done selectively rather than as a routine; otherwise, a greater number of disturbances of the fundus would probably have been found.

Although a condition noted as "stare" was observed in only 6, or 4.4 per cent, of our cases, we desire particularly to call attention to a peculiar fixed, purposeless stare which we have observed in a number of patients with meningitis caused by different organisms. This sign has drawn our attention to a more scrupulous investigation of the patient from a neurologic standpoint and has in several instances been the deciding factor in inducing us to perform a diagnostic lumbar puncture.

We have purposely omitted any reference to the other organs of special sense, since most of our cases occurred in infancy, an age when accurate tests for changes in the special senses are too uncertain to record.

MANIFESTATIONS OF INVOLVEMENT OF THE JOINTS

Debré [41] mentioned that swelling of one or more joints is not rare in meningococcus meningitis of infancy. He observed that the joint is swollen and painful. These signs disappear after a few days. In our series of patients, none had involvement of the joints.

CASE 26.—Quite recently, we encountered a swollen, painful elbow joint in an infant, aged 5 months, with petechiae, who had been ill for three weeks. About the eighteenth or nineteenth day of the disease, the swelling of the joint was noted. Specific therapy, both intravenous and intraspinal, was administered on admission to the hospital on the twenty-first day of the disease. The following day the swelling subsided.

CEPHALIC CRY

Statements regarding the diagnostic value of the so-called cephalic cry permeate the literature. We feel that this has been unduly stressed. We have never been aided in making a diagnosis by any type of cry nor in any instance have we been able to distinguish in this disease a cry which differs from that heard in infants or young children ill with extra-meningeal disease.

CLINICAL FORMS OR TYPES

We have previously referred to the variety of symptoms that this disease may present. This has resulted in the classification of many clinical types by different observers who have described some specially emphasized symptom or group of symptoms generally limited to one

of the anatomic systems. Thus, one reads of gastro-intestinal types, pulmonary types, meningitic types or uremic types, tetanic types, apoplectic types, neuralgic types, typhoid types, delirious types, etc.

Such extensive classification according to type is perhaps more applicable to the disease in adults. While it emphasizes the multiplicity of the manifestations of the disease, it confuses rather than aids in diagnosis. We have found it helpful to classify these cases into clinical forms depending on their degree of severity. There are no rigid limits of the boundaries in such a grouping; namely, fulminating, frank, masked and prolonged. We do not distinguish an "abortive type," because all patients in whom the diagnosis is made are treated, and early disappearance of the symptoms may be attributed to the treatment.

Fulminating Type (Malignant, Hyperacute, Explosive).—The fulminating type composes a definite group, and about 1 case in 20 is thus classified (5.8 per cent) in our series. The outstanding features are the extremely sudden and violent onset, with early collapse and death in a few hours. There may never be any evidence of meningeal irritation (in the most rapidly fatal cases). Relaxation and hypotonicity of muscle groups rather than muscular rigidity are present. Ecchymotic and petechial eruptions are common in this form of the disease. Meningococci can be isolated from the blood in a high percentage of cases, and changes in the sensorium usually usher in the attack. The clinical picture here is one of intense toxemia, probably the result of an overwhelming infection. Three of our patients showed the anatomic manifestations of thymicolymphatic constitution at necropsy. Whether hyperplasia of the lymphatic elements is a cause or a result of the infection is debatable. Westenhoffer [59] observed that children with lymphatic constitutions are predisposed to meningococcus meningitis. We have seen thick purulent cerebrospinal fluid from four to six hours after the onset. In other cases, the cerebrospinal fluid has contained only from 200 to 300 cells and large numbers of extracellular meningococci.

The outcome in this group is unfortunate from the standpoint of therapy, as these patients are usually moribund when specific therapy is available. Of two patients seen recently with this type of the disease, one had been referred to the hospital with purpura hemorrhagica for transfusion; in the other, the condition had been diagnosed intussusception because of the violent vomiting and prostration. Immediate examination of the cerebrospinal fluid of all patients who collapse suddenly, and vigorous intravenous therapy in those in whom examination of the cerebrospinal fluid give positive results must be resorted to if these patients are to be saved.

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59. Westenhoffer: Pathologische Anatomie und Infektionsweg bei der Genickstarre, Berl. klin. Wchnschr. **42**:740, 1905.

Frank Type (Usual, Ordinary, Meningitic).—In the frank type, evidence of meningeal disease is clear-cut, and the picture is that of an acute infection with meningeal irritation. The onset is usually abrupt, with fever and vomiting and, in the majority of cases, convulsions. The classic signs of meningitis are present early: muscular rigidity, particularly of the neck, with retraction of the head; hyperirritability or stupor and bulging fontanel in many young infants. Tache cérébrale, Kernig's sign and Brudzinski's sign can usually be elicited with certainty. A thorough physical examination should always lead to recognition of the disease and early treatment in this type of case.

Masked Type (Mild, Type Frustre).—The clinical manifestations in this type are those of a mild infection in which evidences of meningeal irritation are more or less completely masked. Moderate vomiting and a slight fever usually are present, both of which subside early. The appetite is poor; moderate irritability, and a change in disposition which is usually not alarming to the parents or physician are noticed. The tension of the fontanel is normal, and muscular rigidity with stiffness of the neck is not observed unless carefully sought for. Holt stated that this type is most common in sporadic cases and in early life. Approximately 7 per cent of our patients are in this group, all of whom were less than 1 year of age. Four of this type were under observation in the hospital, and frequent physical examinations were made for from four to five days before lumbar puncture was performed and meningitis recognized. (Two of these patients were in our own hospital and two were in another hospital. One of the last was under observation two weeks without lumbar puncture.) Diagnosis of this type of the disease is particularly difficult in the endemic case when there is no special reason to suspect meningitis. Hyperirritability and muscular rigidity, particularly of the neck, must be searched for more carefully, and lumbar puncture must be resorted to more frequently in vague illnesses if early treatment is to be available in these masked endemic cases.

Prolonged Type (Marantic Type, Hydrocephalic, Chronic Basilar).—The prolonged type of the disease is seen late after the subsidence of the acute clinical manifestations. It is the end-result of one or another of the two former types in patients who survive without treatment. This type is not within the scope of this discussion of symptoms in acute meningitis.

LABORATORY OBSERVATIONS

Spinal Fluid.—The diagnosis of meningococcus meningitis can be established with certainty only by examination of the spinal fluid. Lumbar puncture must be resorted to without hesitation in suspected cases. Delay in carrying out this procedure is rarely justifiable. During a three and a half year period at the Babies' Hospital, 1,855 lumbar punc-

tures were performed without a fatality from the procedure. Often it can be completed with less pain and objection by the patient than an unskilful examination of the ear drums. Occasionally, when cerebrospinal fluid cannot be obtained from the lumbar region, cisternal and ventricular punctures must be done and serum administered through these routes. Hospitalization is usually desirable for these procedures but is no sense essential. Occasionally light anesthesia is advisable in doing lumbar puncture in older children.

Bacteriology.—The presence of meningococci in the spinal fluid makes the diagnosis of this disease certain. In 136 cases, we were able to demonstrate the organism by either smear or culture, or both, in 133 cases. The presence of a purulent fluid without any demonstrable bacteria practically always means meningococcus meningitis. Serum should be administered without waiting for further attempts to demonstrate the organism in smear or for the growth to appear in culture. Rarely, an organism can be demonstrated which at first examination cannot be

TABLE 6.—*Incidence of Cells in Spinal Fluid of Four Patients*

Age	Day of Disease	Number of Cells
2½ months	4th	210
4 months	4th	290
19 months	5th	350
20 months*	5th	120

* Fulminating case.

immediately identified as meningococcus, pneumococcus, streptococcus or staphylococcus. Antimeningococcus serum should be given without delay in such cases until the organism is definitely proved not to be meningococcic. The use of polyvalent serums has eliminated the necessity of typing the meningococcus before treatment.

Cytology.—An increased cell count in the cerebrospinal fluid is present almost uniformly. Polymorphonuclear cells usually predominate, generally composing from 80 to 100 per cent of the total number of cells. We have found viable meningococci in the cerebrospinal fluid in four cases in which the cell count of the fluid was less than 500.

Chemistry.—Neither qualitative nor quantitative chemical examination of the spinal fluid is essential nor of any real assistance in the diagnosis of meningitis or in differentiating the pyogenic meningitides. We have previously reported [60] the quantitative chemical examinations of the sugar and chlorides in this disease before and throughout the period of treatment. The sugar in the cerebrospinal fluid is usually markedly reduced to nearly 20 mg. per hundred cubic centimeters, but in a small

60. Caffey, J. P.; McLean, S., and Sullivan, R. C.: Endemic Meningococcus Meningitis, J. A. M. A. **88**:1865 (June 11) 1927.

number of patients with viable meningococci in purulent spinal fluids there was no diminution in the amount of dextrose in the spinal fluid. We believe the increase in sugar in the spinal fluid to normal values during treatment is of considerable favorable prognostic significance in infants older than 6 months. Quantitative determinations of the con-

TABLE 7.—*Symptoms in One Hundred and Thirty-Six Patients in the Acute Stage of the Disease**

Age in Months	Number of Cases	Fever	Vomiting	Drowsiness, Stupor, Coma	Irritability	Convulsions	Retraction of Head	General Spasticity	Spasticity of Extremities	Stiffness of Neck	Strabismus	Nystagmus	Bulging Fontanel	Tremor	Purpura	Refusal of Food
0 to 3	24	15	9	4	8	10	7	0	2	1	0	1	3	1	0	5
3 to 6	29	22	16	6	15	6	6	1	1	3	4	1	3	0	3	2
6 to 9	28	25	16	4	9	10	5	2	0	6	2	0	2	1	0	1
9 to 12	11	6	9	5	1	5	1	2	0	0	1	0	0	0	0	0
12+	44	34	27	21	11	9	7	2	0	8	6	1	0	1	9	1
Total	136	102	77	40	44	40	26	7	3	18	13	3	8	3	12	9
Percentage		75	56.6	29.4	32.8	29.4	19.1	5.1	2.2	13.2	9.5	2.2	2.8	2.2	8.8	6.6

* Two had herpes; 3, diarrhea; 2, bloody stools; 1, headache; 1, a depressed fontanel and 3, hydrocephalus. In 84, or 62.5 per cent, irritability had been noted on the history or on the physical examination at the time of admission to the hospital. In 63, or 46.2 per cent, drowsiness, stupor or coma was present before admission or was noted at the first physical examination in the hospital. In 115, or 84.5 per cent, spasticity of the spine either at the level of the neck or elsewhere, or spasticity of the extremities or generalized spasticity was noted by the parents or at the first examination in the hospital.

TABLE 8.—*Physical Signs in the Acute Stage of the Disease*

Age in Months	Number of Cases	Kernig Sign	Reflexes Exaggerated	Tache Cérébrale	Brudzinski Sign	Opisthotonos	Retraction of Head	General Spasticity	Stiffness of Neck	Spasticity of Extremities	Purpura	Strabismus	Drowsiness, Stupor, Coma	Irritability	Bulging Fontanel	Nystagmus	Tremor
0 to 3	24	10	14	6	8	3	10	2	1	5	1	3	3	7	14	3	1
3 to 6	29	12	18	11	11	4	14	1	9	4	1	5	5	16	16	3	1
6 to 9	28	11	13	11	10	2	12	2	11	4	0	6	6	10	18	1	0
9 to 12	11	4	6	5	4	0	4	0	5	0	0	4	4	6	4	1	0
12+	44	20	30	9	17	2	18	0	18	0	11	5	11	26	2	3	2
Total	136	57	81	42	50	11	58	5	44	13	13	23	29	65	54	11	4
Percentage		41.9	59.5	30.8	36.7	8.0	42.6	3.6	32.3	9.5	9.5	16.9	21.3	47.7	39.7	8.0	2.9

Total cases with muscular rigidity, 96, or 70.5 per cent

centration of chloride in the cerebrospinal fluid did not give valuable diagnostic or prognostic information.

The colloidal gold reaction, freezing point and specific gravity of the cerebrospinal fluid are interesting statistical data which can be obtained in large laboratories, but at present they are not necessary in the diagnosis and treatment of this disease.

Blood.—Cytology: It is generally agreed that a polymorphonuclear leukocytosis is present. The cytology of the blood is of secondary impor-

tance in this disease, and the diagnosis rests on the spinal fluid manifestations regardless of the blood count. As practically all of our examinations of the blood have been made after the first injection of serum, our data in this regard are not applicable to diagnosis.[61]

Bacteriology.—Blood cultures should be made in all cases, as the presence of meningococci in the blood is a valuable collateral symptom. Blood cultures are particularly important in the fulminating cases, and usually organisms can be grown from the blood when they are not demonstrable in the cerebrospinal fluid. We have taken blood cultures only in selected cases and therefore do not have reliable data as to the incidence of positive blood cultures in all types of cases. As mentioned in the paragraph on the eruption, meningococci can be demonstrated in blood films made from the purpuric areas in the skin.

The bacteriologic and pathologic studies were done under the supervision of Dr. Martha Wollstein.

SUMMARY

1. The analysis of the clinical manifestations in 136 patients with endemic meningococcus meningitis, aged from 23 days to 7 years, is reported.

2. Fifty per cent of the patients were admitted to the hospital more than one week after the onset of the disease. In the majority of cases, the meningitis had not been recognized prior to examination in the hospital. Forty-two of seventy-two patients under medical supervision had been observed for more than three days without diagnosis.

3. The disease is not rare in early infancy, since 17.6 per cent of our patients were not over 3 months of age; 67.6 per cent were in the first year of life. The literature indicates that meningococcus meningitis in epidemics among the civil population approximates the age incidence in the endemic form.

4. In our experience, endemic meningitis occurs almost uniformly in districts in which crowding, poverty and poor hygiene are prevalent.

5. Maternal nursing does not favorably influence immunity.

6. Muscular rigidity, especially at the level of the neck, with or without retraction of the head, was present in 84.5 per cent of the cases. This is the most frequent and important single clinical manifestation.

7. The tendon reflexes were increased in 59 per cent of the cases. They are so frequently altered in health and in other diseases of infancy that they are of doubtful value in the diagnosis of meningitis.

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61. In a previous paper (Blood Platelet Counts in Infants and Young Children, Am. J. Dis. Child. **30**:810 [Dec.] 1925), we showed that the platelet count in meningitis of early life is not altered. This is of diagnostic value in differentiating meningitis with purpura from thrombocytopenic purpura.

8. Kernig's sign was present in 41 per cent of the cases; Brudzinski's sign was found in 36 per cent. Both signs are unreliable when present in infants.

9. Unexplained irritability is one of the most valuable signs which will focus attention on the central nervous system. Its incidence was 62.5 per cent. In 63, or 46.2 per cent, of the patients, drowsiness, stupor or coma was noted before lumbar puncture.

10. Bulging fontanel was noted in 56.6 per cent of the patients with an open fontanel. In patients with a protruding fontanel, the presence of meningitis should always be proved or excluded by examination of the cerebrospinal fluid.

11. Convulsions were present in 29.4 per cent of all cases; nearly all occurred at the onset of the disease. Their absence does not exclude the diagnosis. The presence of unexplained convulsions requires a diagnostic lumbar puncture.

12. Eleven per cent of the patients had a hemorrhagic eruption. Patients with such an eruption who are acutely ill and the examination of whose blood (clotting, bleeding time, platelet count, etc.) does not adequately explain the release of blood into the integument require an immediate diagnostic lumbar puncture and blood culture.

13. Herpes occurred in only 2 of our 136 patients, both over 1 year of age.

14. A characteristic temperature curve is not present in meningococcus meningitis. Fever is frequently absent in the active stages of the disease. The temperature is not a reliable indication of the presence of the disease.

15. Vomiting occurred in 56.6 per cent of the patients. Projectile vomiting occurs rarely. That observed in this group of patients did not differ from that of other acute infections.

16. Diarrhea, frequently mentioned as a common symptom of meningococcus meningitis in infancy, occurred rarely in this group of cases (2 per cent). We do not believe that this symptom is of diagnostic value.

17. Tache cérébrale was noted in 30.8 per cent of the patients. It is so frequently present in other conditions of infancy and childhood that its value is minimized in diagnosis.

18. Strabismus, present in 20.5 per cent of the cases, was the most frequent ocular manifestation of the disease.

THE PIRQUET TEST

COMPARISON OF THE SCARIFICATION AND THE PUNCTURE METHODS OF APPLICATION *

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The methods commonly employed in applying the Pirquet test involve either scarifying the skin with a blunt instrument or making a superficial scratch with a needle. Some clinicians apply the tuberculin after the abrasion has been made, and others perform the test through a drop of tuberculin placed on the skin. The tuberculin is usually permitted to dry in the areas on which it is placed. Both of the foregoing methods of performing the test prove satisfactory in detecting the presence or absence of previous tuberculous infection. In applying the Pirquet test to children attending the Lymanhurst School for Tuberculous Children, it was found that the children preferred to have the test applied by making a single puncture through the skin rather than by scarifying the area with a blunt instrument. Since, from the child's point of view, this method was less objectionable, a study was undertaken to determine whether or not the results obtained by the puncture method were as reliable as those obtained by the scarification method. In addition, to obviate the inconvenience of supervising the children until the tuberculin dried on the skin, the effectiveness of the puncture method of applying the cutaneous test and wiping off the tuberculin immediately was compared with the reactions obtained when the scarification technic was employed and the tuberculin was permitted to dry on the skin.

The tests were performed on 281 children attending the Lymanhurst School for Tuberculous Children. In each instance, the forearm was cleansed with ether, and three drops of tuberculin were applied at distances of about 2 inches (5 cm.) apart. The prominal drop was scarified in the usual manner, and a single puncture was made with an ordinary sewing needle, the epithelium being penetrated through the other two drops of tuberculin. The distal drop was wiped off immediately in each instance.

Comparison of the reaction obtained in the 281 children (table 1) permitting the tuberculin to dry on the skin showed that the scarification and the puncture tests were positive in 223 instances and negative in 58. The agreement was 100 per cent. not only in the total

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figures, but also with respect to the individual cases. Thus the 223 children giving the positive tuberculin reaction by the scarification tests were included without exception among the 223 reacting positively to the puncture test; in other words, of 281 children, the 223 having a positive Pirquet reaction when the tuberculin was applied by scarification were without exception the same patients who reacted positively when tested by the puncture method. It will be noted that since two puncture tests were made on each child, a total of 446 positive reactions was obtained, which agreed with the reactions obtained by the scarification method. This observation demonstrates that the puncture method of applying the Pirquet test is equal in effectiveness to the scarification method.

When the degree or size of the positive reactions was compared, certain differences were noted. Thus, as may be seen from table 1, the size of the reactions in the 223 children was equal in 51 cases, larger in the areas scarified in 84 instances, and larger in 88 children when the puncture method was employed. The positive reactions

TABLE 1.—*Comparison of Cutaneous Reactions to Tuberculin When Applied by Scarification and Puncture Methods*

Number of Cases	Positive Pirquet	Negative Pirquet	Reactions Larger by Scarification	Reactions Larger by Puncture Method	Scarifications and Puncture Tests Equal	Agreement, per Cent
281	223	58	84	88	51	100

obtained, therefore, by a single puncture of the skin through a drop of tuberculin were in general as strong and as definite as those obtained by scarification.

It was noted in performing the puncture tests that when the same needle was used to penetrate two drops of tuberculin on separate areas of the forearm, the area punctured second frequently gave the larger reaction. Apparently, the small amount of tuberculin adhering to the tip of the needle point is influential in producing a greater reaction when penetrating the skin through a second drop of tuberculin. This point, therefore, must be taken into consideration in determining the effect on the reaction of wiping the tuberculin off immediately after the test has been performed. In order to study this observation, two tests were made on each of 131 children, one needle being used for each child (table 2). The area punctured first was wiped off in fifty-two instances, and in seventy-nine cases the drop punctured second was wiped off. The first forty-two children in the former group in which the tuberculin was wiped off immediately from the area punctured had positive reactions, and ten had negative reactions. Here again the agreement with the scarification test was 100 per cent, for the same forty-two had positive

Pirquet reactions; however, the reactions obtained by the puncture method in this group were equal in nine cases, larger in nine cases and smaller in twenty-four cases when compared with the scarification tests. Although making a single puncture through the epithelium with a clean needle and immediately wiping the tuberculin off did not fail in a single instance to detect the positive tuberculin reactions, the reactions obtained were on the average less pronounced than when the scarification technic was employed and the tuberculin was permitted to dry on the skin.

Among the seventy-nine cases in which the drop punctured second was wiped off (table 2), both the scarification and the puncture tests were positive in sixty-nine cases, agreeing individually in this respect in 100 per cent of the cases. The reactions from the puncture tests were equal in fifteen cases, larger in thirty-five cases and smaller in nineteen instances when compared with the size of the reactions obtained by the scarification method. The results indicated, therefore, that although the

TABLE 2.—*Comparison of the Cutaneous Reactions to Tuberculin When Applied by the Scarification and Puncture Method; the Tuberculin Being Wiped Off Immediately from the Site of the Puncture*

Number of Cases	Scarification and Puncture Tests Equal	Reactions Larger by Scarification	Reactions Larger by Puncture Method	Negative Pirquet	Agreement, per Cent	Comment
52	9	24	9	10	100	Drop punctured first wiped off
79	15	19	35	10	100	Drop punctured second wiped off

tuberculin is wiped off immediately from the area punctured second, the reactions obtained are as certain and as pronounced as when the scarification method is used, which permits the tuberculin to dry on the skin.

In a small group of cases, a puncture was made through the skin without first applying a drop of tuberculin to the skin. The only tuberculin introduced in these children was that adhering to the tip of the needle after a puncture had been made through a drop of tuberculin on a different area on the forearm. The reactions in this group were positive in twelve instances by scarification, and by puncture in nine instances. Apparently, the amount of tuberculin introduced by this method failed to give a positive reaction in certain instances.

CONCLUSION

The present study demonstrates that an effective method of applying the Pirquet test is to make a single puncture with an ordinary sewing needle and to penetrate the epithelium through a drop of tuberculin. This method of applying the test has certain advantages in that it may

be performed rapidly and is less objectionable to the child than the scarification technic usually employed. In addition, this method obviates the inconvenience of supervising the child until the tuberculin dries on the skin, for, when the puncture method is employed, the tuberculin may be wiped off immediately without appreciably altering the effectiveness of the test.

SUMMARY

1. The reactions obtained through performing the Pirquet test by making a single puncture with an ordinary sewing needle penetrating the epithelium through a drop of tuberculin placed on the skin were positive in 100 per cent of the 223 children who had had positive reactions when the test was performed by the usual scarification technic.

2. When the same needle is used in making the puncture through two separate drops of tuberculin placed on the forearm, the site punctured second frequently produces the more pronounced reaction.

3. The test did not fail to react in any of the 223 cases in which the drop of tuberculin was wiped off immediately after the puncture was made.

When the test is performed without applying a drop of tuberculin to the skin and when the only tuberculin introduced is that adhering to the tip of the needle after having penetrated through a drop of tuberculin placed on a different area, the reactions are usually but not always positive in persons sensitive to tuberculin.

THE EFFECT OF PARATHYROID EXTRACT ON MINERAL METABOLISM IN INFANTILE TETANY *

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The activity of parathyroid extract in raising the blood calcium suggests other physiologic effects on metabolism. Collip [1] and others, the latest being Hoag, Rivkin, Berliner and Weigele,[2] have reported the treatment in cases of infantile tetany and other forms of tetany by the use of parathyroid extract, controlling their studies primarily by the measurement of blood calcium and phosphorus. Hoag and others also present data on calcium and phosphorus metabolism in two cases of infantile tetany.

It was our purpose not only to study cases of infantile tetany clinically, but also to note the blood serum calcium and inorganic phosphorus, the electrical reactions and the complete mineral metabolism.

METHODS

This paper represents a study made in 1925 on two infants admitted to the New Haven Hospital with infantile tetany and rickets. The children were placed in the metabolism ward under the care of a nurse specially trained in metabolic studies, and the methods previously described [3] were closely followed. The methods of analysis were also the same, with slight improvements, as those described elsewhere.[3] The blood serum calcium was determined according to Clark and Collip's modification of the Kramer-Tisdall method.[4] The inorganic phosphate was analyzed by Brigg's method.[5] The first patient was

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2. Hoag, L. A.; Rivkin, H.; Weigele, C. E., and Berliner, F.: Effect of Potent Parathyroid Extract of Calcium Balance in Infants, Am. J. Dis. Child. **33**:910 (June) 1927.

3. Shohl, A. T., and Sato, A.: Acid Base Metabolism: I. Determination of Base Balance, J. Biol. Chem. **58**:235 (Nov.) 1923.

4. Clark, E. P., and Collip, J. B.: A Study of the Tisdall Method for the Determination of Blood Serum Calcium with a Suggested Modification, J. Biol. Chem. **63**:461 (March) 1925.

5. Briggs, A. P.: Some Applications of the Colorimetric Phosphate Method, J. Biol. Chem. **59**:255, 1924.

a girl; therefore an effort was not made to follow the mineral metabolism, as we do not know of any adequate method for quantitative separation of urine and feces. The urine was collected with thymol chloroform as a preservative and combined into twenty-four hour specimens and kept on ice. The stools were marked with carbon. All food was made in the laboratory from a large supply of dried skim milk and cane sugar and analyzed for all the elements studied. The studies of metabolism and other studies were performed without any known error in feeding, collection of specimens or analysis. Vomiting did not occur.

REPORT OF CASES

CASE 1.—A girl, aged 4 months, was admitted to the ward of the New Haven Hospital on Feb. 19, 1925. She had not had any previous illness. She had been breast fed for only two weeks. Cod liver oil had not been given, and she had not been given special exposure to sunshine. The day before admission to the hospital, she had vomited after the early morning feeding. She had a convulsion that evening and one every two or three hours afterward, and once after admission to the hospital. The results of examination were practically negative except that she was found to have a temperature of from 39 to 40 C. (102.2 to 104 F.), rapid breathing and a bulging left ear drum, which was incised. The roentgen-ray examination showed rickets at the wrists. Chvostek's sign was positive. Carpopedal spasm was not noted. The electrical reactions showed: cathodal closing contraction, 2; anodal closing contraction, 3.5; anodal opening contraction, 3, and cathodal opening contraction, 4. The blood serum showed 5.3 mg. per hundred cubic centimeters of calcium, 9.3 mg. of phosphorus and 3.6 mg. of calcium in the cerebrospinal fluid. These observations are typical of tetany—low electrical reactions and a low calcium and a high phosphorus content. Without other treatment, the child was given subcutaneously 1 cc. of parathyroid extract-Collip[6] at 4 p. m. and a second similar dose at 8. At 10, the electrical reactions were: cathodal closing contraction, 2; anodal closing contraction, 4.5; anodal opening contraction, 3, and cathodal opening contraction, 11. Blood taken the next morning showed 5.35 mg. of calcium per hundred cubic centimeters of blood serum. While we waited for the report on the blood, 1 cc. of parathyroid extract-Collip was given only in the afternoon, lest an overdose be given. The next day, three doses of 1 cc. of parathyroid extract-Collip were given. The day following, two doses of 2 cc. each, were given; the day afterward, 12 cc., and the next day, 6 cc., which exhausted our supply. At this time, the temperature had become normal. The electrical reactions remained high, but still showed anodal reversibility. The Chvostek sign was absent, and the patient did not have convulsions. The calcium did not rise above 5.7 mg., but the phosphorus had made a significant drop to 3.1. At this point, treatments with calcium chloride orally and by ultraviolet light were given, and the patient made an uneventful recovery. On discharge from the hospital, March 9, the calcium was 11.3 mg. per hundred cubic centimeters and the phosphorus, 7.5 mg. This patient received 22 cc. of parathyroid extract-Collip in six days, which, while not enough to raise the calcium content, caused a drop in the phosphorus, and raised the electrical reactions and presented convulsions. The danger of an overdose and lack of more material prevented the administration of larger doses of the extract.

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6. The parathyroid extract was generously supplied us by the Eli Lily Company; it contained 5 units per cubic centimeter.

Comment.—Except for the slight effect on the electrical reactions and the absence of progressive symptoms, benefits from the treatment were not noted. The dosage was very small.

CASE 2.—A boy, aged 6 months, weighing 7,220 Gm., was admitted to the New Haven Hospital on March 16, 1925. The child had never been ill previously, had been breast fed for one month, and then fed on diluted whole milk and water mixtures. Cod liver oil had not been given, and the social worker reported the family as being dirty and uncooperative. The mother could not be induced to give the child sun baths. Four days before admission, the child had two convulsions which lasted from thirty minutes to one hour each. On the day of admission, there was a convulsion lasting fifteen minutes. The results of the physical examination were essentially negative, except for slight enlargement of the costochondral junctions and at the wrists. The roentgen-ray examinations showed mild rickets. The Chvostek sign was positive. There was not any evidence of intercurrent infection. The electrical reactions were: cathodal closing contraction, 0.5; anodal closing contraction, 2.5; anodal opening contraction, 1.5, and cathodal closing titanic contraction, 0.5. The blood serum calcium was 6.7, and the phosphorus, 6.7 mg. Examination of the blood revealed: white blood cells, 21,000; hemoglobin, 70 per cent (Sahli), and red blood cells, 5,984,000. The smear was normal. The Wassermann reaction was negative. An analysis of the urine did not show any abnormality. The cerebrospinal fluid was negative for globulin and contained 3 cells. The blood p_H was 7.39, and the carbon dioxide content, 56 per cent by volume. Cerebrospinal fluid p_H was 7.33, the carbon dioxide content, 53 per cent by volume, and the calcium, 4.4 mg. per hundred cubic centimeters. The daily food in the hospital was equivalent to 1 liter of skim milk plus 100 Gm. of cane sugar, 110 calories per kilogram. The first dose of parathyroid extract-Collip was given subcutaneously at 6 p. m. The child was put on the metabolism bed the next morning. He was given 1 cc. of parathyroid extract-Collip every four hours until seventeen doses had been given, then four doses of 2 cc. At the end of four days, he was removed from the metabolism bed, and the marked stool was recovered. The second day afterward he was put back on the metabolism frame for three days, being fed on the same diet without further treatment. When he was removed after the second period, he was again given 1 cc. of parathyroid extract-Collip every four hours for nine doses.

The data concerning the calcium and the phosphorus in the serum and the electrical reactions are given in table 1. The only other notable result is that the urine which was acid on admission became alkaline, p_H 8.6, on the third day of administration of parathyroid extract. It became acid, p_H 6.6, when the treatment was interrupted, and again became alkaline during the second course of parathyroid extract. At this point, the experiment was stopped; the child was given ultraviolet light and cod liver oil, and had an uneventful recovery. He was discharged one week later.

COMMENT

In this uncomplicated case of infantile tetany, the salient data are given in table 1 and in the chart. The action of the parathyroid extract-Collip in raising the calcium and lowering the phosphate in the serum is unmistakable. The electrical reactions showed lessened galvanic neuromuscular irritability. This, in agreement with the observations of Hoag

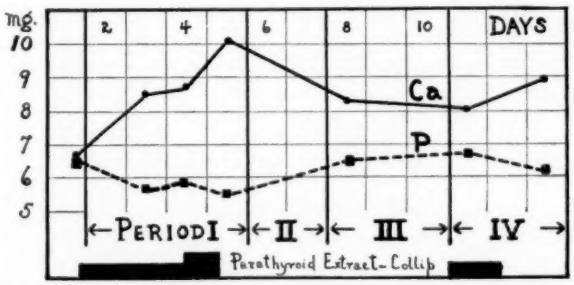
and others,[2] is only a temporary symptomatic cure for tetany. When the drug was withheld, the serum calcium and phosphorus and the electrical reactions to a less extent reverted to their former values. The urine became alkaline under treatment. The second course of parathyroid treatment produced the same effects as the first.

The result in case 1 is scarcely a fair test of the action of the drug, as the available supply was so limited. Although an effect on the blood

TABLE 1.—*Data on Calcium, Phosphorus and Electrical Reactions in Case 1*

Date of Entrance	Serum Calcium Mg. Percentage	Serum Inorganic Phosphorus Mg. Percentage	Electrical Reactions in Milliamperes CC.	AC.	AO.	CO.	CCT	
3/16/25	6.7	6.7	0.5	2.5	1.5	...	5.0	Period I Parathyroid extract-Collip, 25 cc., in 21 doses in 126 hours; on metabolism bed
3/17/25	...	...	2.0	5.5	3.0	>5.0		
3/18/25	8.5	5.6	3.0	10.0	5.0	...	13.0	
3/19/25	8.9	5.8						
3/20/25	10.0	5.7	5.0	7.0	2.0	...	15.0	
								Period II Rest period; off bed
3/23/25	8.3	6.5	5.0	7.0	2.0	...	15.0	Period III No medication; on metabolism bed
3/24/25	...	...	3.0	9.0	2.0	14.0		
3/25/25	...	...	3.5	8.0	5.0	>16.0		
								Period IV Parathyroid extract-Collip, 9 cc., in 9 doses in 54 hours; off bed
3/26/25	8.1	6.7	2.5	9.8	8.5	8.0		
3/28/25	9.0	6.3	5.0	6.0	4.0	...	>15.0	

In this table CC. indicates cathodal closing contraction; AC., anodal closing contraction; AO., anodal opening contraction; CO., cathodal opening contraction; CCT., cathodal closing tetanic contraction.



The calcium and inorganic phosphorus of the blood serum during parathyroid therapy.

calcium was not noted, the inorganic phosphorus in the serum, was considerably lowered and the galvanic irritability was lessened. Hoag and others [2] state: "The reaction of individual patients to parathyroid extract varies tremendously and depends on factors which cannot at present be evaluated."

When parathyroid extract was first given, the metabolism of all inorganic acids and bases was noted in the food, urine and feces during

periods I and III, the first while parathyroid was being administered and the second following its omission. Data are given in tables 2 and 3. The values for a normal infant of about the same age, on the same diet, studied at the same time, are given for comparison.

TABLE 2.—*The Mineral Metabolism (Case 2) in Infantile Tetany* *

		Feces: Period†			Urine: Period			Total: Period			Balance: Period		
	Food, Gm.	I Gm.	III Gm.	Normal Gm.	I Gm.	III Gm.	Normal Gm.	I Gm.	III Gm.	Normal Gm.	I Gm.	III Gm.	Normal Gm.
Ca	1.555	1.435	1.435	1.103	0.003	0.005	0.012	1.438	1.440	1.115	0.117	0.115	0.440
Mg	0.114	0.12	0.11	0.113	0	0	0	0.12	0.11	0.113	—	—	—
Na	0.697	0.036	0.030	0.05	0.562	1.01	0.550	0.598	1.04	0.600	+0.10	0.343	0.097
K	1.96	0.087	0.054	0.10	1.575	0.680	1.650	1.662	0.734	1.75	+0.30	+1.23	0.21
P	1.325	0.574	0.66	0.47	0.418	0.476	0.590	0.992	1.136	1.06	0.333	0.190	0.265
S	0.48	.....	.....	.....	0.331	0.353	0.37	.....	.....	.....	0.149	0.127	0.11
Cl	1.56	0.08	0.12	0.10	1.23	1.27	1.35	1.31	1.39	1.45	0.25	0.17	0.11

* The values are expressed in grams of each element per twenty-four hours.

† Period I indicates the preiod during the administration of parathyroid extract; period III began two days after the last dose of parathyroid extract. No medication was given. In the normal period, the patient received the same amount and kind of food as in case 2.

TABLE 3.—*The Mineral Metabolism (Case 2) of Infantile Tetany* *

		Feces: Period			Urine: Period			Total: Period			Balance: Period		
	Food, Cc. 0.1 N	I Cc. 0.1 N	III Cc. 0.1 N	Normal Cc. 0.1 N	I Cc. 0.1 N	III Cc. 0.1 N	Normal Cc. 0.1 N	I Cc. 0.1 N	III Cc. 0.1 N	Normal Cc. 0.1 N	I Cc. 0.1 N	III Cc. 0.1 N	Normal Cc. 0.1 N
Ca.....	774	702	702	501	1	2	6	703	704	507	71	70	267
Mg.....	95	96	94	93	0	0	0	96	94	93	—1	+1	2
Na.....	302	17	13	32	244	440	239	261	453	271	41	—151	31
K.....	500	22	14	27	404	174	423	426	188	450	74	312	50
(A) Bases —	1671	837	823	653	649	616	668	1486	1439	1321	185	232	350
PO_4.....	770	335	384	273	242	276	324	577	660	597	193	110	173
SO_4.....	297	...	...	...	174	189	204	174	189	204	123	108	93
Cl.....	440	23	34	28	348	358	380	371	392	408	69	48	32
(B) Acids +	1507	358	418	301	764	823	908	1122	1241	1209	385	266	298
Excess (A — B)...	—164	—479	—405	—352	+115	+207	+240	—364	—198	—112			
Base balance (A — B)...	...	...	...	...	...	...	...	...	...	...	+200 Neg.	+34 Neg.	—52 Pos.

* The values are expressed in cubic centimeters of tenth-normal solution of each element per twenty-four hours.

Base is indicated by —; acid by +.

The results show clearly, in agreement with Hoag and others,[2] that balance of calcium and phosphorus is not negative during these two periods. In both the experimental and after-period, the calcium and phosphorus are greater in the stool than in the normal control, and the retention of calcium is less than that of the normal child. That parathyroid extract produces a negative calcium balance has been shown by

Greenwald and Gross [7] during experiments on dogs with the parathyroids removed, and by Hunter and Aub [8] in patients with lead poisoning. The acid-base balance of the body is altered during or following the administration of parathyroid extract. The urine becomes less acid and the stool more alkaline. An acid retention, instead of a base retention, results. During the administration of parathyroid extract, a normal amount of potassium and sodium was excreted in the urine; in the after-period, an excess of sodium was excreted, which resulted in a negative sodium balance, and so little potassium was present in the urine that a large amount was retained. Gamble, Ross and Tisdall,[9] who studied the sodium and potassium metabolism in a case of tetany in which the patient was treated by calcium chloride, found that the acid which increased the excretion of all the other bases caused a slight lowering of the amount of potassium in the urine. This observation also indicates some abnormality of potassium metabolism in tetany. If it is assumed that the effects noted were produced by the cure of tetany, this would mean that tetany is associated with a retention of potassium, which is in exact opposition to Greenwald's [10] sodium theory.

CONCLUSIONS

Small amounts of parathyroid extract were administered in two cases of infantile tetany. The drug had little effect on one infant. The second case showed a control of the symptoms and an improvement as measured by clinical and chemical results. This improvement is transitory and disappears when treatment is stopped. The alteration is brought about without gross changes in the calcium and phosphorus metabolism. During the period of administration of parathyroid extract with relief of symptoms, there was a normal excretion of sodium and potassium. During the relapse following withdrawal of parathyroid extract, the excretion of sodium increased, and potassium was retained.

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7. Greenwald, I., and Gross, J.: The Effect of the Administration of a Potent Parathyroid Extract Upon the Excretion of Nitrogen, Phosphorus, Calcium and Magnesium; Solubility of Calcium Phosphate in Serum and in the Pathogenesis of Tetany, J. Biol. Chem. **66**:217 (Nov.) 1925.

8. Hunter, D., and Aub, J. C.: Lead Studies: XV. The Effect of the Parathyroid Hormone on the Excretion of Lead and of Calcium in Patients Suffering from Lead Poisoning, Quart. J. Med. **20**:123 (Jan.) 1927.

9. Gamble, J. L.; Ross, G. S., and Tisdall, F. F.: Studies in Tetany: I. The Effect of Calcium Chlorid Ingestion on the Acid-Base Metabolism of Infants, Am. J. Dis. Child. **25**:455 (June) 1923.

10. Greenwald, I.: The Supposed Relation Between Alkalosis and Tetany, J. Biol. Chem. **54**:285 (Oct.) 1922.

THE HEARTS OF NORMAL CHILDREN

I. CLINICAL STUDIES, INCLUDING NOTES ON EFFORT SYNDROME *

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A school offers a much better opportunity than any clinic for the study of normal children. It is more feasible to follow a given group of school children over a number of years than the same number of children in a clinic, and it is much easier to determine abnormal circulatory behavior in a school child under standard conditions than in the environment of a clinic. Especially is this true of a private school of the so-called "experimental" type where great opportunity is given to observe children in a natural environment. For this reason, the Bureau of Educational Experiments has conducted a series of studies on growth in children in the City and Country School of New York City. As physician to the Bureau, I had the opportunity of acting as physician in this school from 1919 to 1926 when the records used for these studies were being collected. At the beginning of this period a health program was organized, the function of which was not only to prevent epidemics of contagious disease and to detect serious defects in the children, but also to minimize the occurrence of preventable diseases and defects. Each year, complete physical examination with the addition of examination of the stool and urinalyses were made for every child; hemoglobin estimations were also made when deemed necessary. The younger children were reexamined in the spring of each year, and reexaminations of the circulatory system were made during the year whenever a child returned to school after a serious illness. All children were weighed and measured every two months during the school year. Full medical and physical histories of the children were obtained and careful follow-up work was done by a trained social worker. Nine hundred and ninety-three yearly examinations on 325 children from 2 through 13 years are represented in this study. Many of the same children were followed for years, and from four to seven successive yearly physical examinations were made on a group of 118. As part of these studies on the development of the heart and circulatory system in childhood, a teleoroentgenogram and an electrocardiogram were obtained each year of as many children as possible.

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* From the Bureau of Educational Experiments.

It was hoped that during the long periods in which the same children would be under observation, it would be possible to collect valuable data on so-called "precardiac" signs. Either by chance, or possibly because of the preventive health program, none of the children developed organic heart disease during the period of observation. Four children with recognized organic heart lesions were taken in as new pupils. The records of these children are of course not included in the following survey, and the records of one boy obviously suffering from a granular dyscrasia associated with hypertension have also been omitted. In addition to the compilation of normal standards, special studies have been made of the group of children in whom murmurs were heard at some time, and of another group that had symptoms referable to the circulatory system.

STATISTICAL METHODS

In a review of the literature concerning the circulatory system in children, there are many tabulations of averages, but articles in which accurate quantitative methods have been applied to the statistics with a view to establishing standards of normal deviations from the average are few. And yet it is obvious that in studying a child, it is difficult to recognize the first signs of pathologic change in the circulation unless the normal limits of variation as well as the average or mean figures are known. The range of distribution of physiologic measurements on normal persons can be calculated by Pearson's method of calculating the standard deviation or measure of deviation from the mean or average.[1] By this means it is possible to indicate the normal range of variability, and it is obvious that the nearer the data gathered on an individual are to the average of all normal persons, the greater the chances that the measurements on the individual are normal; conversely, when the measurements begin to approach the extreme limits of the standard deviations, one is more likely to be dealing with a pathologic state. This is particularly true of heart measurements, in which small differences are significant and the actual variability is low. Kilgore[2] has emphasized the potential value of this method for the solution of medical problems. The percentage errors for some of the following statistics have been calculated, and since the possible error in calculation has been found very small—much less, for instance, than would equal 1 mm. of mercury in making a reading of the blood pressure or a tenth of a centimeter in measuring a teleoroentgenogram—they have been omitted from the statistical tables in order to simplify them. All ages are calculated to the nearest birthday. The cases are not evenly distributed. The greater number are fairly evenly divided in the age groups from 4 through 10 years. However, the statistics without any smoothing or redistribution give fairly smooth curves, so that our figures for the smaller groups—ages 2 and 3, and 12 and 13—are presented, although I realize that further work needs to be done on these age groups before final standards can be made.

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1. The standard deviation is the square root of the arithmetic mean of the square of the deviations from the average of distribution.

2. Kilgore, E. S.: Relation of Quantitative Methods to the Advance of Medical Science, J. A. M. A. **75**:86 (July 10) 1920.

Normal standards for recumbent and vertical heart rate and for systolic and diastolic blood pressure and pulse pressure have been calculated. The general conditions are excellent for obtaining these data. The physician's office is situated in the school building adjacent to the classrooms. The children are scarcely ever hesitant about coming for examination, and only occasionally does a new child fear the procedure. Practically all examinations have been made in the morning between 10 and 12 o'clock. All blood pressure readings were taken by the auscultatory method with the child in a sitting position with the arm horizontal, and all readings were made on the left arm. The cuff used was 9.5 cm. wide. Systolic pressure was read at the first distinct continuous sound; diastolic pressure at the last loud sound. Practically all observations of the blood pressure were made

TABLE 1.—*Average Measurements of Horizontal and Vertical Pulse Rates with Standard Deviations*

Age	Boys			Girls		
	Number of Cases	Average Pulse Rate	± s.d.	Number of Cases	Average Pulse Rate	± s.d.
		Horizontal				
2	5	108	13.8	5	95	2.6
3	17	95	9.9	16	96	8.9
4	37	92	8.1	38	91	7.8
5	43	88	7.4	47	91	8.3
6	55	86	8.7	46	90	7.8
7	46	85	10.9	45	87	6.6
8	47	81	9.0	38	83	9.2
9	47	82	9.9	33	84	7.4
10	31	79	6.8	30	84	6.8
11	27	80	7.8	27	80	7.9
12	23	79	10.0	26	83	10.4
13	8	79	10.7	5	74	7.8
		Vertical				
2	11	108	15.1	13	114	15.2
3	18	102	6.6	14	103	7.6
4	31	100	12.9	34	99	11.6
5	42	95	12.3	43	99	12.0
6	51	95	11.0	45	94	9.9
7	40	92	10.6	42	94	9.8
8	42	90	11.2	32	92	13.7
9	41	88	9.3	24	90	9.5
10	24	89	11.6	26	89	9.5
11	17	85	8.7	16	91	10.3
12	12	82	10.1	10	89	9.0
13	..	..	....	4	82	5.4

three times in succession, and if the first reading showed a striking difference from the two succeeding, as occasionally happened, it was discarded, a fourth reading made, and the average recorded. Heart rate was counted for fifteen seconds, and the result was multiplied by 4 except when marked arrhythmia was present, in which case a full minute was counted.

HEART RATE

In the series, as shown in table 1, there is a slightly more rapid horizontal heart rate in girls from 5 through 10 years than in boys of the same ages, the difference being from two to five beats a minute, with an average of three beats a minute. The vertical pulse rates are practically equal for boys and girls from 3 through 10 years, except at the age of 5. At 11 and 12, the rates for girls are decidedly higher than

for boys, probably showing the greater instability of the circulatory system which appears with adolescence. Thus a sex difference is found in horizontal pulse rate but not in vertical pulse rate, except at puberty. There have been many reports on the normal heart rate in children, and all have emphasized the great variation in rate in children of all ages. It has been difficult to compare these figures with others, as in most cases it is not stated whether the author has recorded the recumbent or vertical pulse rate or both, and in some cases differentiation between the sexes has not been made. If the average horizontal and vertical pulse rates for boys and girls is combined, one secures a table of averages in children which is practically identical with many published tables. This failure to take both position and sex into account may well be the reason why the literature on rate of pulse in children is full of diverse

TABLE 2.—*Average Pulse Rate in Children Considering Position and Sex*

Age	Boys*	Girls*	Boys and Girls†
2	108	104	106
3	99	100	99
4	98	95	96
5	92	95	93
6	91	92	91
7	94	91	92
8	86	88	87
9	85	87	86
10	84	87	85
11	83	86	84
12	81	86	83
13	.. ..	78	..

* Combining horizontal and vertical pulse rates.
† Combining sexes as well as horizontal and vertical pulse rates.

opinions. According to Trousseau,[3] the pulse is more rapid in girls after the age of 6 months, whereas Vierodt [4] said that the pulse is of equal frequency in boys and in girls until 5 years of age. Gundobin [5] claimed that the size of the child is the greatest factor affecting the frequency of the pulse rate, and since, on the whole, boys are larger than girls, their pulse rates are slower. Further studies on the pulse rate in children should take sex, position and size into account in any attempt to formulate standards.

BLOOD PRESSURE

As shown in table 3, practically no sex difference in systolic blood pressure at the ages given could be demonstrated, and the differences at 3 and 5 years are not apparently significant. The diastolic pressure

3. Trousseau, quoted by Feldman: Ante-Natal and Post-Natal Child Physiology, New York, Longmans, Green & Company, 1920, p. 432.

4. Vierodt, H.: Anatomische physiologische und physikalische Daten und Tabeller, Jena, 1906.

5. Gundobin, N.: Die Besonderheiten des Kindesalters, Berlin, 1912.

also does not show any significant sex differences, contrary to the observations of Faber and James.[6] Michael[7] found that sex did not influence blood pressure, provided height and weight were the same in both sexes. A study of blood pressure in relation to height showed only the steady increase to be expected with advancing age. The pulse pressure also does not show any sex differences, again contrary to the observations of Faber and James. The statistics for the systolic blood pressure in children older than 6 accord very well with the figures given by these same writers in their careful study, while the figures for the younger

TABLE 3.—*Average Measurements of Systolic and Diastolic Blood Pressure with Standard Deviations*

	Systolic Blood Pressure					
	Boys			Girls		
Age	Number of Cases	Systolic	± s.d.	Number of Cases	Systolic	± s.d.
3	14	93	3.8	12	89	6.9
4	35	94	6.4	40	93	6.7
5	49	98	9.0	54	95	5.8
6	57	98	8.2	49	97	7.9
7	54	98	6.7	47	99	7.5
8	48	100	5.2	41	100	6.2
9	48	101	5.1	37	101	8.2
10	30	105	7.2	34	104	10.9
11	28	104	8.8	29	105	7.8
12	23	102	12.4	27	104	11.5
13	11	105	5.7	7	105	11.2
	Diastolic Blood Pressure					
	Boys			Girls		
	Number of Cases	Diastolic	± s.d.	Number of Cases	Diastolic	± s.d.
3	14	66	6.7	12	67	4.7
4	35	67	5.4	40	66	5.0
5	49	70	8.2	54	67	5.1
6	57	70	4.9	49	69	4.8
7	54	70	5.7	47	70	5.9
8	48	73	6.0	41	71	6.1
9	48	72	5.5	37	71	6.3
10	30	75	8.0	34	74	6.1
11	28	73	6.9	29	74	6.3
12	23	73	9.4	27	75	4.1
13	11	75	4.9	7	74	9.4

groups are uniformly higher. It is possible that the results obtained in this study agree closely with theirs for the older groups, because in both series healthy school children were examined, whereas the younger age groups of Faber and James may include the 150 children from the clinic who were "apparently well" and who were added to their group of school children. The figures for diastolic blood pressure are higher than those of Faber and James, and the standard deviations in the

6. Faber, H. K., and James, C. A.: The Range and Distribution of Blood Pressures in Normal Children, Am. J. Dis. Child. **22**:7 (July) 1921.

7. Michael, M.: A Study of Blood Pressure in Normal Children, Am. J. Dis. Child. **1**:272 (April) 1911.

figures are on the whole greater than those given by these writers. The figures in the present study are not corrected for ideal distribution, and smoothing of curves has not been done, so that the results are not absolutely comparable. It seemed wise not to obliterate any evidence of the marked normal variability of blood pressure readings in children, which is lost sight of if so much work is done in trying to obtain smooth curves.

SIZE OF HEART

The data relating to the size of the heart by percussion and position of the apex beat and of the point of maximal impulse of the heart in this study agree closely with those of Seham[8] in his recent review of the literature. They are not included in this study, first, because, they did not seem as well suited to treatment by the statistical method as the other measurements and, second, because in a subsequent article the normal variation in heart measurements as shown by the more objective measurements of the teleoroentgenograms will be given.

TABLE 4.—*Average Measurements of Pulse Pressure for Both Sexes*

Age	Boys	Girls
3	27	22
4	27	27
5	28	28
6	28	28
7	28	29
8	27	29
9	29	30
10	30	30
11	31	31
12	29	29
13	30	31

EXERCISE TEST

For the first four years of this study, an exercise test was used regularly to measure the increased rate of the pulse after exercise and to time the return of the pulse to normal. It was felt to be of little clinical significance except in the few cases of effort syndrome, and when Barringer[9] demonstrated that similar tests were worthless in measuring cardiac efficiency and his results were confirmed by Wilson,[10] the tests were discontinued. Instead, a modified exercise test with repeated readings of the blood pressure as described by Wilson was substituted in some cases.

8. Seham, M., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 242.

9. Barringer, T. B., Jr.: Studies of the Heart's Functional Capacity, Arch. Int. Med. **20**:829 (Dec.) 1917.

10. Wilson, May G.: The Circulatory Reactions to Graduated Exercise in Normal Children, Am. J. Dis. Child. **20**:188 (Sept.) 1920.

SINUS ARRHYTHMIA

Sinus arrhythmia was commonly found during physical examinations of this group of children. The frequency did not vary directly with age, although, on the whole, it occurred with more regularity in the younger children. Arrhythmia was present on the average in 86 per cent of all patients, practically without any difference due to sex in its incidence. A great deal of this arrythmia was of the respiratory type. Not only were irregularities on the whole less common as the child grew older, but they became less marked. Thus in boys the percentage showing marked arrhythmia dropped steadily from 46 per cent at 2 years to 17 per cent at 12 years, and the girls showed a similar decline. This was disconcerting to find in view of the fact that in the electrocardiograms, as will be shown later, the incidence of arrhythmia increases with age in boys and girls, the average occurrence being only 62 per cent—much less than the percentage obtained from physical examination. The only reasonable explanation of this apparent discrepancy, which will be discussed more fully in the section on electrocardiography, is that the children became more or less frightened by the procedure of taking an electrocardiogram; consequently, their heart rates were increased and they breathed more quickly, thus decreasing the incidence of both sinus and respiratory arrhythmia. Friberger [11] studied 321 unselected children, from 5 to 14 years old, and on examination found some respiratory arrhythmia in all; this was confirmed by sphygmographic curves. Hecht [12] found cardiac irregularity due to respiration practically universal in children; from birth to puberty gradually it becomes more marked.

PULMONIC SECOND SOUND

An accentuation of the pulmonic second sound was present so frequently in this group of normal children, and especially in the group of children with nonorganic heart murmurs, that it is difficult to believe that its occurrence can be of help in the early diagnosis of organic cardiac disease. In the great majority of the children, the pulmonic second sound was louder than the aortic second sound, although there is a gradual decrease with age in the percentage of cases showing this condition. In boys at 12 and in girls at 11, it again becomes increasingly more common for the pulmonic second sound to be louder than the aortic, so that the figures for 12 years are about the same as those for early childhood (chart 1).

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11. Friberger, R.: Ueber Arhythmie bei gesunden Kindern, Arch. f. Kinderh. **58**:30, 1912.

12. Hecht, A. F.: Der Mechanismus des Herzaktion im Kindersalter, Ergebn. d. Inn. Med. u. Kinderh. **11**:324, 1913.

About 10 per cent of all the pulmonic second sounds were accentuated. In a group of children with nonpathologic murmurs, 64 per cent showed an accentuation of the pulmonic second sound, which was marked in half of the cases. Among the eleven children with true growth hypertrophy, who will be fully described in the section on roentgen-ray examinations of the heart, only the average percentage showed an accentuation of the second pulmonic sound. It is interesting to note that an attempt to correlate prominence of the pulmonary artery in the roentgenograms with an accentuation of the pulmonic second sound was a failure, as only the usual percentage of roentgenograms showing prominent pulmonary arteries occurred in those cases. When the pulmonic second sound was not louder than the aortic second, the sounds were

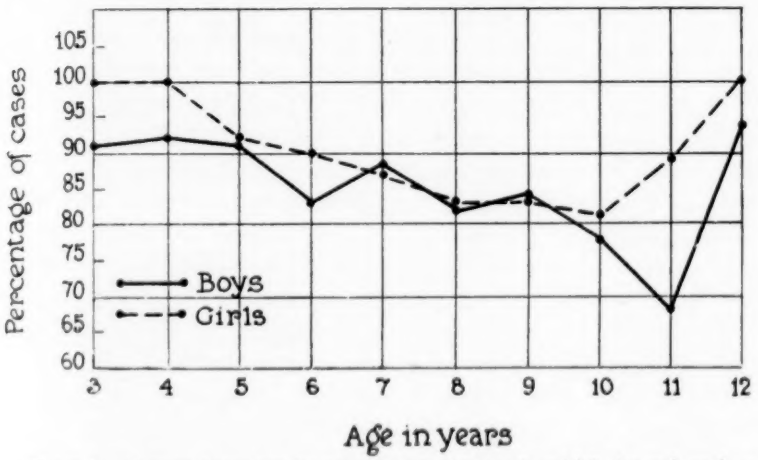

Chart 1.—Curves showing the percentage of cases in which the pulmonic second sound is louder than the aortic second sound.

usually about equal in intensity. Three per cent of girls of 9 years of age showed a louder aortic second sound than pulmonic, and this was the largest percentage found in any age group. Before 6 years of age, the aortic second sound was never louder than the pulmonic. Accentuated aortic second sounds were not heard.

MURMURS

The literature is so full of discussions of murmurs, their classifications and causes, that this subject is approached with some hesitation. There were twenty-seven children in the total group of 325 in whom murmurs were heard at some time, and, since it is certain by subsequent history and examinations that all these murmurs were nonpathologic, it seems worth while to give a more detailed description of the cases.

Five of the children were followed for only one school year; of the twenty-two others observed for longer periods, twelve presented a murmur once and none thereafter. The remaining ten children showed a murmur on two or more occasions. A murmur was not noted constantly in any child although one anemic, malnourished girl had a murmur in five of seven examinations.

Thirteen of the patients were boys and fourteen girls, and the condition was discovered in the great majority before they were 7 years of age, the greatest number falling in the fifth and sixth years. Hochsinger [13] found "nonpathologic" murmurs most common between

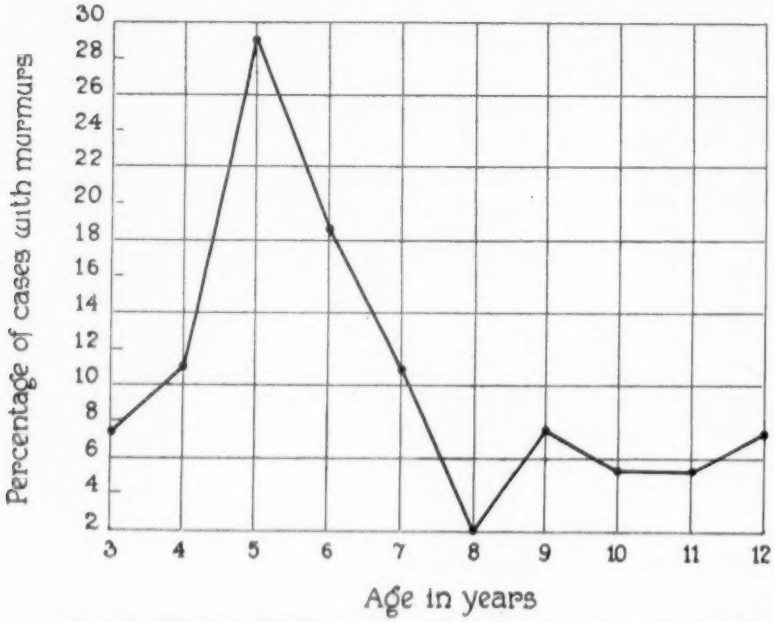

Chart 2.—Diagram showing ages at which murmurs first appeared in the children under observation.

the ages of 10 and 14, and Seham [14] said they become more frequent from 6 years to puberty.

The time of the murmurs was invariably systolic, and the intensity varied from very faint to loud, often changing markedly in the same child with short intervals between examinations. Most of the murmurs were blowing, and many had a decided musical quality. The duration varied greatly but was most frequently short.

13. Hochsinger, C.: Ueber bedeutungslose Geräusche in der Präcordialgegend von Kinderhn und Jugendlichen, Arch. f. Kinderh. **60**:377, 1913.

14. Seham, M., in Abt: Pediatrics, vol. 4, p. 251.

The murmur was usually confined to the apex, but was occasionally heard over the entire precordium. In the majority of cases, it was increased by exercise, but in two cases, it disappeared following exercise. The murmur was not influenced in any way except occasionally in intensity when the position was altered. A murmur localized to the pulmonic area was heard only twice, transiently in a girl of 7 who was just beginning to show a growth hypertrophy on the roentgenogram and in a girl of 5 who was not examined subsequently. In the latter case, the murmur was apparently a true cardiopulmonary one, as it disappeared entirely when she held her breath. With this single exception, it seemed impossible to arrange the murmurs according to any of the numerous classifications, and therefore they have been called simply nonpathologic or accidental, since none of the children developed organic disease.

A constantly aberrant pulse rate or blood pressure was not noted in these children, although transient records of high or low blood pressure and of rapid pulse rate were common. On the whole, each one showed the amount of sinus arrhythmia average for age and sex, but, as mentioned before, a high percentage of these cases showed an accentuation of the second pulmonic sound. There were no points common to most of the electrocardiograms that were abnormal. All the various types of hearts were present on the roentgenograms, and the pulmonary artery was not enlarged in more than the normal percentage of cases. None of the children with murmurs showed true microcardia on roentgen-ray study, but two of them had murmurs during the first year, when they showed true growth hypertrophy on the teleoroentgenograms. Four of the children who showed transient murmurs had symptoms referable to the circulatory system, part of the picture of effort syndrome. No abnormal observations were common to all or to most of this group of children with murmurs, except possibly the marked frequency of an accentuation of the second pulmonic sound. In most cases, only time made absolutely certain the diagnosis of nonpathologic murmurs.

SYMPTOMS

Murmurs were of doubtful clinical significance in the cases in which they occurred, but a group of cases in which the symptoms presented were referable to the circulatory system have been most interesting from a clinical standpoint. The most common of these were cyanosis, pallor or dyspnea on exertion and undue fatigue. The group of children presenting these symptoms became well known to the school physician, because the teachers and the nurse frequently sought advice concerning them.

One of these children showed a true small heart from 7 until 10 years of age, his transverse heart measurements being well below the lower limit of normal on all examinations. Blood pressure and pulse rate were close to the average. During the last three years under observation the size of his heart came within the normal limits for the first time; with this acceleration in the rate of cardiac growth, the record of his symptoms ceased.

Four girls showing true growth hypertrophy also had symptoms which called the attention of the observers to the possibility of cardiac disease. They showed pallor and fatigue on slight exertion during the first years in which the diagnosis of enlarged heart was made evident by the teleoroentgenogram. After a few years, although their hearts remained large, the two younger girls adjusted themselves to this condition, and the records of their symptoms ceased. The two older girls continued to have signs of vasomotor instability, evidenced by frequent fainting attacks, until puberty was established.

EFFORT SYNDROME

During the war, there occurred a great recrudescence of interest in a set of symptoms variously known as the effort syndrome, the soldier's heart or the irritable heart. All writers on this subject have emphasized the long histories of vasomotor instability and fatigue which characterize this condition. Many believe that this group of symptoms is due to a congenital weakness.

There were five children in the school group studied whose lack of capacity for sustained effort, either mental or physical, together with symptoms referable to the circulatory system, seemed to make them, if not patients with true cases of effort syndrome, at least candidates for this diagnosis in later years. All these children were intelligent, but rather apathetic, preferring to be onlookers rather than participants, especially in outdoor games or other situations involving much output of energy. The youngest child whose condition was diagnosed as an effort syndrome was 4 and the oldest 9. There were three boys and two girls in the group. The symptoms in these children were not constant. Frequently weeks or months would pass without complaints from the teacher or mother, and then, following an infection or unaccustomed physical exertion, symptoms would again be apparent. Intensification of symptoms after infection was noted in adults by Meakins and Gunson.[15] Fatigue was the most common symptom, whereas it was probably least common in the group of children previously discussed who had true microcardia or growth hypertrophy. Dyspnea on slight physical exertion

15. Meakins, J. C., and Gunson, E. B.: The Pulse Rate After a Simple Test Exercise in Cases of "Irritable Heart," Heart **6**:285 (May) 1917.

was also frequently a symptom, but cyanosis or pallor were rarely noted, except by the physician, again in contrast to the other group in which pallor was a symptom often noted by the teachers.

These five children all looked fairly robust; one of the girls was only of average height, but the others were above normal, and all were of normal weight. None of these children had good muscle tone, and all had poor posture of the typical fatigue type. The records of these children are full of notes concerning the character of their heart sounds. A tendency to embryocardia was frequently noted, and impure first sounds at the apex were often heard. Marked accentuation of the pulmonic second sound was common, and all except one child showed transient murmurs. There was a marked tendency to rapid pulse rate, both horizontal and vertical, and the pulse frequently remained high after exercise. This was especially true of the children in whom exercise tests induced symptoms such as dyspnea or cyanosis. Meakins and Gunson also found this true of adults. The blood pressure was frequently well below the average. There was a delayed rise in blood pressure following exercise tests.

None of these cases showed any common characteristics or abnormalities in the electrocardiograms, except that S waves in the first lead were almost always present and were deeper than normal. Since this is a common observation in the hearts of young children, it may suggest some delay in development in the hearts of children showing effort syndrome.

The transverse width of the heart as shown on teleoroentgenograms was below the average in most cases, although it remained within the limits of the standard deviations. On first examination, the three most marked cases were 1 cm. below average heart size and the other two cases, 0.3 cm. Meakins and Gunson [16] found in cases in adults that the hearts in those with effort syndrome are on the average somewhat smaller than normal (0.7 cm. in orthodiagraphic tracings).

In the first of these cases which occurred in the school and which happened to be the most marked, the child was treated as a potential cardiac patient, but when a second case occurred and I recognized that it was probably a juvenile case of effort syndrome, a change in policy was made. The children were encouraged to increase gradually the amount of exercise they were taking; their families and teachers were advised to ignore circulatory symptoms as far as possible and to try to instil in the minds of these children a liking for outdoor games and sports. Graded exercises were impossible because of the type of school, but rhythmic dancing classes were part of the curriculum. Before they

16. Meakins, J. C., and Gunson, E. B.: Orthodiagraphic Observations on the Size of the Heart in Cases of So-Called "Irritable Heart," Heart **7**:1, 1918-1920.

passed from observation, four of these five children had hearts of average size. Physical examinations did not reveal any symptoms or deviations from the normal. In the fifth patient, aged 12 years, the heart was still below average, and a true adolescent effort syndrome seemed to be developing.

If this condition is congenital, the patients probably cannot be cured, and no one knows how the four children who no longer have symptoms will meet difficulties in the future; it has seemed worth while, however, to try to prepare these children in school to accustom themselves to sustained effort, both mental and physical.

CONCLUSIONS

1. Girls from 4 to 12 years of age have a more rapid horizontal heart rate than boys of the same age.

2. The vertical pulse rate is equal in boys and girls, except at 11 and 12 years of age, at which time the heart rate of the girls is higher than that of the boys.

3. Significant sex difference in blood pressure in children from 3 through 13 years of age could not be demonstrated.

4. Pulse pressure does not vary greatly with sex and only slightly with age.

5. In contrast to the electrocardiographic observations on the same children, irregularities of the heart decrease with age in children from 2 through 13 years old.

6. Transient systolic murmurs were present in 6.6 per cent of the cases, but were not of demonstrable significance.

7. In a group of 325 normal school children, symptoms referable to the circulatory system were present only in those showing marked deviations from the normal heart size or in those showing the "effort syndrome."

GROWTH STANDARDS OF HEIGHT AND WEIGHT FOR GIRLS IN PRIVATE SCHOOLS *

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CHARLOTTE GOWER, M.A.
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Although the best standards to be used for public school work are those published by Baldwin, we believe that separate tables are necessary for measurements of pupils in private schools. For boys, such tables have already been published.[1] This paper presents data on 1,030 girls in five private school in or near Chicago: Alcott School (73), Chicago Latin School for Girls (192), North Shore Country Day School (139), Francis W. Parker School (85) and the University of Chicago Laboratory School (541). Part of the measurements were

TABLE 1.—*Height for Age in Girls in Private Schools*

Age Nearest Year	Number Measured	Height, Net	
		Cm.	Inches
5	11	112.8	44.4
6	35	118.1	46.5
7	58	123.4	48.6
8	84	128.3	50.5
9	106	133.2	52.4
10	104	138.0	54.3
11	115	143.6	56.5
12	111	149.2	58.7
13	98	155.0	61.0
14	90	158.6	62.4
15	81	161.3	63.5
16	61	162.2	63.9
17	49	163.0	64.2
18	22	163.4	64.3
19	4	163.9	64.5

made in the first four schools by one of us (C. G.) and part by Mrs. Naomi Giffen and in the last named school by the school physician. The principal points in the technic were as follows: the height was measured with the subject barefoot and against a wall, and the weight was taken on balance scales with the subject naked (occasionally with underwear, the weight of which was allowed for before a record was made). The time of measurement varied from about 9 a. m. to 2 p. m., being made, when possible, during the morning, two or more hours after breakfast.

* Received for publication, Dec. 3, 1927.

* This is one series of articles by members of the staff of the Behavior Research Fund, Illinois Institute for Juvenile Research, Chicago, Herman M. Adler, M.D., Director, Series B, No. 119.

1. Gray, H., and Fraley, F.: Growth Standards for Private School Boys, Am. J. Dis. Child. **32**:554 (Oct.) 1926.

TABLE 2.—*Weight for Height in Girls in Private Schools*

Metric		English	
Height, Net	Weight, Net	Height, Net	Weight, Net
103	16.7	41	38
104	17.0	41½	38
105	17.3	42	39
106	17.7	42½	40
107	18.0	43	41
108	18.3	43½	42
109	18.7	44	43
110	19.1	44½	44
111	19.6	45	45
112	20.0	45½	46
113	20.4	46	48
114	20.8	46½	49
115	21.3	47	50
116	21.7	47½	51
117	22.2	48	52
118	22.6	48½	53
119	23.0	49	55
120	23.4	49½	57
121	23.8	50	58
122	24.2	50½	59
123	24.6	51	60
124	25.1	51½	62
125	25.6	52	63
126	26.0	52½	64
127	26.5	53	66
128	27.0	53½	68
129	27.5	54	70
130	28.0	54½	72
131	28.5	55	74
132	29.0	55½	76
133	29.5	56	78
134	30.1	56½	80
135	30.7	57	82
136	31.3	57½	84
137	31.9	58	87
138	32.5	58½	89
139	33.2	59	91
140	33.8	59½	94
141	34.6	60	97
142	35.1	60½	100
143	35.8	61	102
144	36.7	61½	105
145	37.5	62	107
146	38.4	62½	110
147	39.2	63	112
148	40.1	63½	115
149	41.1	64	117
150	42.0	64½	120
151	43.0	65	123
152	43.9	65½	124
153	44.9	66	127
154	46.0	66½	129
155	47.1	67	131
156	48.3	67½	133
157	49.4	68	134
158	50.5	68½	136
159	51.2	69	137
160	51.9	69½	138
161	52.6	70	139
162	53.3		
163	54.0		
164	54.7		
165	55.3		
166	56.0		
167	56.6		
168	57.5		
169	58.3		
170	59.0		
171	59.6		
172	60.3		
173	60.9		
174	61.4		
175	61.8		
176	62.3		
177	62.7		
178	63.2		

Compared with figures for some other children, our girls were: (1) definitely taller than the well known Baldwin-Wood medians;[2] the excess, averaging the differences for all ages, was 20 mm. (0.8 inch) and ranged from 6 (age 14) to 33 mm. (age 6). (2) They were taller by a greater margin than the Francis W. Parker and University of Chicago School girls reported by Baldwin in 1914.[3] This difference is interesting, because pupils from those same two schools composed 61 per cent of our series. (3) They were slightly shorter than the 323 girls from Eastern private schools whose measurements were plotted by Benedict and Talbot.[4]

To find the average height for a girl of a given age in a private school one looks in table 1; to find the average weight for a girl of that stature, one refers to table 2.

Our series is small, and we are enlarging it, but judging from our experience in successive previous papers on boys reporting progressively larger series, and by the closeness of the points plotted for actual means to the smoothed curves, we do not expect the averages to be much altered. The values in the present tables were read from the smoothed curves.

In the current continuation of the work, we are also taking account of racial stock and of other body measurements. Both these considerations will, we believe, afford a further improvement in accuracy of weight prediction.

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2. Baldwin, B. T.: Personal communication giving the median stature for age in the children reported by him and by Dr. T. D. Wood in another form.

3. Baldwin, B. T.: Physical Growth and School Progress, Bull. 10, U. S. Bur. Education, 1914, p. 22.

4. Benedict, F. G., and Talbot, F. B.: Metabolism and Growth, Publ. 302, Carnegie Inst., Washington, 1921, pp. 44 and 46, and personal communication to the authors.

Case Reports

CELIAC DISEASE*

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Celiac disease is now recognized as a clinical entity, and since its modern description, dating from 1888,[1] much has been written about it without any great light being thrown on its etiology. Bacterial infection has presumably been the cause, according to some investigators, but no one has produced the disease with these offending organisms, nor have they always been found in well defined cases in the hands of other competent investigators.

Indigestion of one type or another, that is, indigestion caused by fat or by carbohydrate, but particularly by fat, has been advanced as the cause of the disease, largely on the basis of what is apparent in the stools of these children. The mineral salts have also been suspected of causing this condition. Postmortem examination has not thrown much light on the etiology.

The following unbiased study was made on a patient with celiac disease in order to record such facts as could be demonstrated under trying conditions. Observations made on only one patient are recorded; this seems justifiable since the condition is not common, and cooperation on the part of the patient is necessary in carrying out the procedure recorded.

REPORT OF CASE

B. L. H., a girl, born Aug. 1, 1922, was bottle fed excessively on carbohydrates until she was about 1 year of age when she was given her first variation in diet from the milk and carbohydrate formula. She teethed normally and seemed to thrive, according to the mother, until April, 1924. Then, for no apparent reasons, she began to refuse her food and became listless and waxy in appearance. Her weight of 28 pounds (12.7 Kg.) was rapidly dissipated; her legs became spindly and her abdomen distended; one month later, she had a profuse diarrhea. From then on her stools were typical of celiac disease, and she had attacks with remissions until I first saw her in January, 1925. At this time she was 2 years and 5 months old and weighed 12 pounds (5.4 Kg.) She was extremely dehydrated, had a subnormal temperature, and passed twelve or more porridge-like stools a day; her chest was full of moist râles. Just before I saw her, her diet had consisted exclusively of buttermilk.

My primary effort was made to save the child's life. Intraperitoneal injections of Ringer's solution were given; water was taken by mouth and retained; necessary stimulation was administered, and thick arrowroot gruel was given for three days, which effectually checked the diarrhea. Well cooked spinach and

* Received for publication, Oct. 3, 1927.

1. Gee: St. Bartholomew's Hosp. Rep. **34**:17, 1888.

carrots, with gelatin, were added to the diet; soft egg and diaprotein biscuits were then given. This diet was followed by beef juice; skimmed milk, given sparingly at first, then in larger quantities, was added, and the diet was made fairly liberal. The child gained, recovered its ability to talk and walk, and by August, 1925, had regained its high water mark in weight, 28 pounds, its weight at the onset of the illness. All this, however, did not occur without periods of remission and loss of weight, so that a verification of the diagnosis of celiac disease made after the proper studies had been completed following the child's recovery from the critical stage in which I found it was justified.

During September, 1925, the child had a severe loss of weight and an increase in the number of stools, so that I determined on the following observations as a possible means of discovering just what was happening so far as this child was able to cooperate with me. She had achlorhydria. Nothing else of note was found in the contents of the stomach. I found that she would be cooperative in the washing of the stomach, so I decided to pass a duodenal catheter.

My plan was first to study the enzymes recovered, then the effect of acid peptone and cane sugar after a period of starvation. The next step was the administration of 20 per cent cream, a determination of the blood cholesterol being made before and after the fat meal. Charcoal was administered with the cream, and all stools were studied.

TABLE 1.—*Contents of Samples Drawn from Duodenal Catheter*

	Sample 1	Sample 2
Lipase	70	63
Trypsin	54	37
Amylase	1.19%	1.16%
Bile	+	+

DIARY

Tuesday, Sept. 22, 1925:

12 m.: A duodenal tube was introduced into the stomach.

2:30 p. m.: The fluoroscope showed the tip 2 inches below the pylorus.

4:30 p. m.: Sample collected, representing steady drip for two hours (sample 1).

4:45 p. m.: Sample 2 collected.

The contents of the two samples are shown in table 1.

Supper was given, and the nourishment was continued until midnight.

Wednesday, Sept. 23, 1925:

10:00 a. m.: Sample A drawn from tube (breakfast had not been given).

10:25 a. m.: Fifty cubic centimeters of 1 per cent acid peptone plus 2.5 Gm. cane sugar given.

10:30 a. m.: Sample B 1 discarded.

10:40 a. m.: Sample B 2 showed marked difference in turbidity (increase).

10:45 a. m.: Sample B 3.

10:55 a. m.: Sample B 4 discarded.

11:10 a. m. Sample B 5, clear yellow.

11:30 a. m.: Five cubic centimeters of 20 per cent cream given by mouth.

11:45 a. m.: Sample C 1.

12 m.: Sample C 2.

12:15 p. m.: Sample C 3.

The total fat values in table 3 are rather high normals for a child on a fat-free diet, but the figures show an increase after the fat meal.

TABLE 2.—*Examination of Samples*

Exhibit A	Lipase*		Trypsin†	Amylase‡
	With Olive Oil	With Cream		
Sample A	50	55	50	0.64%
Sample B 2	37	53	19	0.0%
Sample B 3	34	24	7	0.0%
Sample B 5	39	92	65	0.20%
Sampel C 1	85	89	56	0.52%
Sample C 2	91	27	66	0.98%
Sample C 3	77	50	14	0.0%

* Results represent acidity produced by 100 cc. of duodenal contents on 1 Gm. of fat (olive oil, cream) expressed in terms of tenth normal sodium hydroxide.

† Results represent acidity produced by 100 cc. of duodenal contents on 1 Gm. of gelatin, expressed in terms of tenth normal sodium hydroxide.

‡ Results represent percentage of sugar in solution after 1 cc. of duodenal contents has acted on 1 Gm. of starch for one hour.

TABLE 3.—*Examination of Blood Before and After Administration of Fat*

Exhibit B

	Blood Cholesterol
Before fat meal	145 mg. per 100 cc. of blood
Four hours after fat meal (cream)	160 mg. per 100 cc. of blood
Normal values	140–170 mg. per 100 cc. of blood

	Total Blood Fat (minus cholesterol)
Before fat meal	581 mg. per 100 cc. of blood
Four hours after fat meal	705 mg. per 100 cc. of blood
Normal blood fat in percentage, 0.1 to 0.7	100–700 mg. per 100 cc. of blood

TABLE 4.—*Reports of Stools*

	Test, per Cent	Duplicate, per Cent	Average, per Cent
Specimen 1:			
Total fat	36.76	34.50	35.63
Neutral fat	30.10	24.45	27.27
Soaps and combined fatty acids	.....	.....	8.36
Specimen 2: (5 cc. of cream was given, and charcoal was administered before this specimen was passed)			
Total fat	32.88	32.72	32.80
Neutral fat	19.30	16.70	18.00
Soaps and combined fatty acids	.....	.....	14.80
Specimen 3:			
Total fat	39.04	35.20	37.12
Neutral fat	18.90	15.90	17.40
Soaps and combined fatty acids	.....	.....	19.72
Specimen 4:			
Total fat	58.56	56.64	57.60
Neutral fat	18.50	14.80	16.15
Soaps and combined fatty acids	.....	.....	41.45
Specimen 5:			
Total fat	51.68	52.20	51.94
Neutral fat	19.20	23.12	21.16
Soaps and combined fatty acids	.....	.....	30.78
Specimen 6: (charcoal was given before this specimen was passed at the same time that 5 cc. of 20 per cent cream was given; charcoal was present in this specimen)			
Total fat	36.96	36.72	36.84
Neutral fat	25.50	21.20	23.35
Soaps and combined fatty acids	.....	.....	13.49
Specimen 7:			
Total fat	48.42	49.02	48.72
Neutral fat	22.60	23.84	22.72
Soaps and combined fatty acids	.....	.....	21.50
Specimen 8:			
Total fat	54.26	53.74	54.00
Neutral fat	28.22	27.50	27.86
Soaps and combined fatty acids	.....	.....	26.14

Because of the controversy that arises immediately on the presentation of any studies of enzymes from the standpoint of the relative merits of the possible methods that might be employed, I found a child, aged 2½ years, weighing 30 pounds (13.6 Kg.), with no digestive disturbance, who would also cooperate with me. Tables 5 and 6 show the results of this experiment.

BACTERIOLOGIC STUDIES AND BLOOD CHEMISTRY

Periodic examination of the stools of the child with celiac disease for two years showed that *Bacillus coli-communis* was the predominating organism. Occasionally, *Bacillus bifidus* was recovered. The recovery of the latter organism, however, was not necessarily coincident with a recurrence of symptoms. I never recovered *Bacillus infantilis*. Cultures from the duodenal contents did not show any growth.

TABLE 5.—*Analysis of Contents of Stomach and Duodenum in Normal Child*

Analysis of Contents of Stomach				
Hydrochloric Acid	Total Acidity			Combined
0	12			37
Analysis of Duodenal Contents				
	Fasting Contents			
	Trypsin	Lipase	Amylase	
Exhibit AA	59	71	1	
	After Peptone Was Given			
	Trypsin	Lipase	Amylase	Color
1	9	7	0	Pea green, turbid
2	23	4	0	Bluish green, less turbid
3	28	1	0	Green
4	46	28	0.99%	Light green
5	29	36	1.35%	Dark green
	After 5 cc. of Cream Was Given			
1	81	46	0	Brownish green
2	59	55	1.60%	Light green with curds
3	47	12	1.92%	Light green, many curds

The values for blood fat have already been recorded. The blood sugar was normal. Blood calcium and other blood chemical determinations were not made.

COMMENT

The tables demonstrate the following facts:

1. Bile, lipase, trypsin and amylase are present in the duodenal content, which confirms the observation of Taylor.[2] These enzymes are present in the same amounts as in the presumably normal child in variations as to both fasting time and feeding time. That they have the same digestive power is also demonstrated, it being noted particularly, however, that animal fat is probably digested more readily than vegetable fat.

2. Taylor, Rood: Tr. Am. Pediat. Soc. **34**:100, 1922.

TABLE 6.—*Examination of Blood and Stools of Normal Child*

Exhibit BB

Blood

Blood fat, 666 mg. per 100 cc. of blood 4 hours after fat meal

Stools	Per Cent
Specimen 1: (fat-free diet)	
Total fat	23.40
Neutral fat	7.00
Soaps and combined fatty acids	16.40
Specimen 2: (after 5 cc. of cream was given)	
Total fat	28.40
Neutral fat	14.00
Soaps and combined fatty acids	14.40
Specimen 3: (fat-free diet)	
Total fat	25.85
Neutral fat	18.14
Soaps and combined fatty acids	17.71
Specimen 4: (after 5 cc. of cream was given)	
Total fat	22.86
Neutral fat	10.70
Soaps and combined fatty acids	12.16
Specimen 5: (fat-free diet)	
Total fat	29.00
Neutral fat	16.72
Soaps and combined fatty acids	14.28

TABLE 7.—*Comparative Study of Duodenal Contents of Child with Celiac Disease*

		Normal Child	Child with Celiac Disease	
			Sample 1	Sample 2
Fasting Contents	Lipase	71	70	63
	Trypsin	59	54	37
	Amylase	1	1.19	1.16
	Bile	+	+	+

* For the duodenal contents after peptone and cream were given, exhibits A and AA should be compared. A comparison of the examination of the blood, four hours after ingestion of fat, is shown in exhibits B and BB.

TABLE 8.—*Comparison of Stools of Normal Child and Child with Celiac Disease*

	Normal Child:	Child with Celiac Disease:
	Per Cent	Per Cent
Specimen 1: (fat-free diet)		
Total fat	23.40	35.63
Neutral fat	7.00	27.27
Soaps and combined fatty acids	16.40	8.26
Specimen 2: (after 5 cc. of cream)		
Total fat	28.40	38.80
Neutral fat	14.00	18.00
Soaps and combined fatty acids	14.40	14.80
Specimen 3: (fat-free diet)		
Total fat	25.85	37.12
Neutral fat	18.14	17.40
Soaps and combined fatty acids	17.71	19.72
Specimen 4: (after 5 cc. of cream)		
Total fat	22.86	57.60
Neutral fat	10.70	16.60
Soaps and combined fatty acids	12.16	41.00
Specimen 5: (fat-free diet)		
Total fat	29.00	51.94
Neutral fat	16.72	21.16
Soaps and combined fatty acids	14.28	30.78
Specimen 6: (after charcoal and 5 cc. of cream)		
Total fat	.....	36.84
Neutral fat	.....	23.35
Soaps and combined fatty acids	.....	13.49
Specimen 7: (charcoal present)		
Total fat	.....	48.72
Neutral fat	.....	22.72
Soaps and combined fatty acids	.....	26.00
Specimen 8:		
Total fat	.....	54.00
Neutral fat	.....	27.86
Soaps and combined fatty acids	.....	25.14

2. The fats in the stools of patients with celiac disease are greatly increased and are much greater than the intake. In the normal child, the output of fat is greater than the intake, but the difference is not as marked as in the child with celiac disease. This is also evident from the fact that the stools of the normal child will have a relatively high fat content when the child is on a fat-free diet, while, under the same circumstances, those of a child with celiac disease will have an excessive fat content.

3. The absorption of fat seems to be as readily attained experimentally in the child with celiac disease as in the normal child; the same holds true of carbohydrates. Clinically, there seems to be no reason for questioning protein metabolism.

These tests seem to point to the accuracy of the statement of Hill and Bloor [3]:

> When moderate amounts of fat are fed, the fat of the feces is largely independent of the diet and in composition approaches that from a fat free diet.
>
> The comparative constancy of composition of the feces fat favors the idea of a fat excretion from the intestine but while an excretion is probable it cannot be regarded as proven in view of the undetermined influence of lipoid from free cellular material.
>
> The feces fat cannot ordinarily be regarded as unabsorbed food fat, and therefore feeding experiments as a test of the extent of utilization of food fat are of doubtful value unless account be taken of the amount and kind of fat which appears in the feces independently of the food.

These tests seem to point also to the accuracy of the statement of Sperry and Bloor [4]:

> The composition of the food fat is different from that of the feces fat.
>
> There is a marked similarity between the blood and fecal lipoid with regard to the ratios of solid to liquid fatty acids and the melting points of the nonvolatile fatty acid fraction and its solid component, which makes it probable that the fatty material of the feces has its origin largely in the blood.

The etiology of celiac disease must be sought in some agent or influence that disturbs that side of the metabolic balance which is concerned with the transfer of used fats and carbohydrates back again into the intestinal tract.

It is only fair to say that, in opposition to this view, an increased blood fat is held by some to be sufficient reason for an increased excretion of fat. That deduction is not apparent, however, in the foregoing work. Further studies must be made on the possibilities of a bacterial etiology in conjunction with the foregoing type of work, which in itself can be extended. It must, however, be done in the earlier days of the illness, and such a study will be undertaken at an early opportunity.

3. Hill and Bloor: Biol. Chem. **53**:171, 1922.
4. Sperry and Bloor: J. Biol. Chem. **60**:261, 1924.

AURICULAR FIBRILLATION IN CHILDHOOD

REPORT OF CASES *

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Auricular fibrillation is so characteristically a condition of adult life and old age that it is scarcely thought of in the consideration of cardiac diseases in childhood. In order to determine its incidence according to age, Cohn tabulated 434 cases of auricular fibrillation which had been reported by Lewis, Frey and Semerau. His table [1] did not reveal a single case occurring in patients up to the age of 10 years; only 2 per cent of the patients were between the ages of 10 and 20 years. His observations are epitomized in table 1.

TABLE 1.—*Relation of Fibrillation to Age (Based on 434 Cases)*

Age Group	Percentage of Total Cases in Group
0–10	0
10–20	2
20–30	10
30–40	17
40–50	22
50–60	21
60–70	20
70–80	6
80–90	2
	100

A search of the literature reveals very few reports of children exhibiting auricular fibrillation. Resnick and Scott [2] recently reported a case and prefaced their report with a tabulation of all previous instances on record of auricular fibrillation in children 10 years of age or less. Their summary is given as table 2.

REPORT OF CASES FROM THE LITERATURE

In the case reported by Price and MacKenzie, the patient developed complete heart block as well as auricular fibrillation during an attack of diphtheria, the apex rate dropping to 40 beats per minute before death.

Sutherland and Coombs described a case of intensely virulent rheumatic pancarditis in a child, aged 5, which ended fatally in six days. When first seen by

* Received for publication, Nov. 2, 1927.

1. Cohn: Auricular Fibrillation, in Nelson's Loose-Leaf Medicine, New York, Thomas Nelson & Sons, vol. 4, p. 333.

2. Resnick and Scott: Auriculr Fibrillation, Am. J. Dis. Child. **31**:357 (March) 1926.

the authors, the patient was cyanosed, the heart dilated, and the pulse markedly irregular, feeble and of low tension. "Polygraph tracings showed a gross type of pulse irregularity, while the venous tracing from the neck was of the ventricular type, there being no trace of an auricular wave."

Ehrenreich's patient was a girl, aged 9, who had rheumatic heart disease and gave a history of several attacks of decompensation, relieved by rest in bed, restricted diet and digitalis. Physical examination revealed enlargement of the heart with signs of mitral stenosis and regurgitation. Roentgen-ray examination of the chest showed extreme enlargement of the shadow of the heart in all diameters. While the patient was undergoing treatment in bed and digitalization for an attack of decompensation, arrhythmia appeared. Electrocardiographic studies proved the presence of auricular fibrillation.

The patient whose case was reported by Ogden, a boy, aged 10, was given treatment in the hospital for rheumatic heart disease with mitral stenosis and regurgitation and chronic tonsillitis. Shortly after discharge from the hospital, he developed acute otitis media and a gastro-intestinal upset. On reentry the patient had a rapid, irregular pulse with a pulse deficit of 40 and moist râles throughout both lungs. An electrocardiographic tracing showed coarse auricular fibrillation with a ventricular rate of 100 and diphasic T_2 wave. The patient was digitalized and quinidine was administered in increasing doses.

TABLE 2.—*Records of Auricular Fibrillation in Children*

Case	Author	Age	Type of Heart Disease
1	Price and MacKenzie..... Heart **3**: 233, 1911-1912	9	Diphtheritic
2	Sutherland and Coombs..... Heart **5**: 15, 1913-1914	5	Rheumatic
3	Ehrenreich ..... New York M. J. **99**: 269, 1914	9	Rheumatic
4	Ogden ..... Am. J. Dis. Child. **29**: 767, 1925	10	Rheumatic
5	Resnick and Scott..... Am. J. Dis. Child. **31**: 357, 1926	4	Probably rheumatic

Normal rhythm was not restored until the dose had reached 44 grains (2.86 Gm.) per day; the patient having received 270½ grains (17.55 Gm.) in fourteen days. Seven months later auricular fibrillation appeared again, coincident with the development of chronic otitis media. The patient was digitalized this time, but "quinidine was not given in view of the fact that there was so much damage to the heart, because too much of the drug had been needed on the previous stay in the hospital to restore normal rhythm, and because of the likelihood of frequent relapses." The patient was discharged on a maintenance dosage of digitalis.

Resnick and Scott's patient, a boy, aged 4, had a history of cyanosis in early life which was somewhat suggestive of congenital heart disease. At 2½ years he had had a severe sore throat, but the history did not reveal any joint pains, chorea, or other acute illnesses. The patient entered the hospital with decompensation of the heart, edema of the face and lower extremities, marked dyspnea and orthopnea, slight cyanosis of the mucous membranes and enlargement and tenderness of the liver. The tonsils were large. The heart rate was 160, and its rhythm was irregular. An electrocardiographic tracing revealed auricular fibrillation. The patient was digitalized and improved rapidly. After discharge from the hospital, he did fairly well for several months, but died about nine months later, following progressive dyspnea and edema. Necropsy was not performed. The etiology of this patient's cardiac condition was not proved, but the authors were inclined to regard it as acute rheumatic myocarditis.

Resnick and Scott suggested that: "Undoubtedly auricular fibrillation occurs more commonly in young children than one would suspect from the small number of cases which have hitherto been reported. The apparent rarity is probably due, in part at least, to the fact that this type of irregularity is not ordinarily considered in children, and, in the absence of graphic records, is quite naturally confused with sinus arrhythmia, perhaps associated with extrasystoles."

Two instances of auricular fibrillation were encountered in children during the past summer at the Seaside Hospital. We desire to report these cases because of the supposed rarity of the condition in early life and because they would seem to agree with the foregoing statement of Resnick and Scott.

REPORT OF TWO CASES

CASE 1.—John L., aged 9½ years, was admitted to the Seaside Hospital on June 8, 1927, with a diagnosis of chronic rheumatic myocarditis, associated with mitral stenosis and insufficiency. His illness began at the age of 5, when he had rheumatic fever with swollen, painful joints and a "weak heart." He spent three weeks at the Seaside Hospital in the summer of 1923. Physical examination at that time showed a heart enlarged to the left and downward with presystolic and systolic murmurs typical of mitral stenosis and insufficiency. He was classed as a IIA cardiac, i. e., a patient with an organic lesion necessitating only slightly diminished physical activity. He was treated with rest, heliotherapy and graduated exercises and was discharged improved. Soon afterward he had a severe setback as the result of a fright and was in a hospital for several months, followed by nine months in a convalescent home for persons with cardiac disease. His tonsils were removed at this time. He was fairly well until February, 1926, when he had an attack of measles which disabled him for a month. He then went back to school until February, 1927. From that date until his present admission, he had been feeling "too sick and weak to attend school or to play very much." He had attended the Bellevue cardiac clinic for three months prior to admission at the Seaside Hospital, and had received digitalis.

Physical examination on admission revealed a pale, weak, poorly nourished child of good mentality. There was some dyspnea and orthopnea, but no marked edema. The left side of the thorax bulged considerably, and the cardiac impulse was diffuse and heaving. The heart was enlarged to the left, the apex impulse being 8 cm. from the midsternal line and in the sixth interspace. The heart was totally irregular, without discernible rhythm. The apex rate was 122 and the radial rate 98, making a pulse deficit of 24 beats. There was a systolic thrill. A loud, harsh, systolic murmur was heard all over the precordium, but was loudest at the apex. A harsh diastolic murmur was also noted in the mitral and tricuspid regions, but with no presystolic accentuation. Roentgen-ray examination showed considerable cardiac enlargement, especially marked in the region of the left auricle. The total transverse diameter of the heart was 11.9 cm., and the internal diameter of the chest, 20 cm.

A diagnosis of auricular fibrillation was made, and the patient was given 30 minims (1.9 cc.) of tincture of digitalis three times daily. The apex rate rapidly diminished to from 60 to 70, the pulse became stronger and more regular, and the deficit disappeared. The patient was allowed out of bed and was placed on a maintenance ration of 0.1 Gm. of digitalis daily. Precordial pain occasionally necessitated the administration of codeine to secure sleep at night.

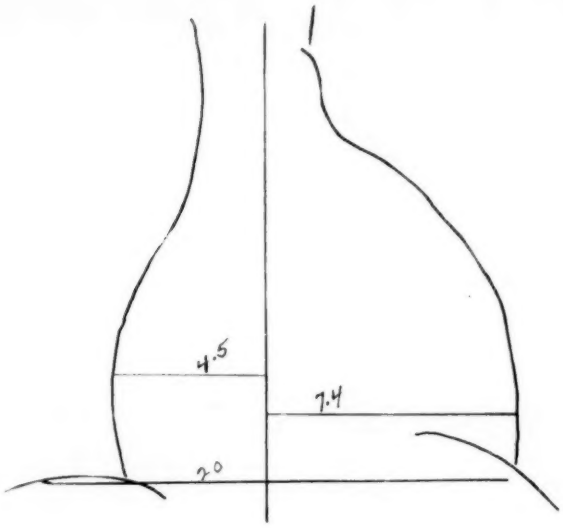

Fig. 1.—Film of the heart at six feet shows a marked enlargement of the shadow of the heart in the region of the left auricle, suggesting mitral disease.

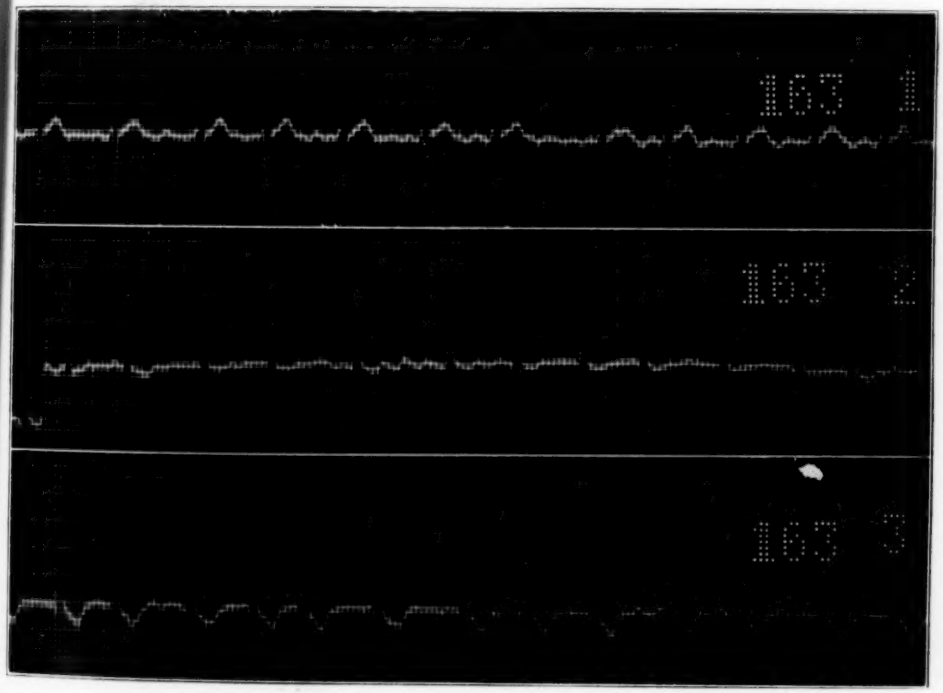

Fig. 2.—Electrocardiogram of J. L., showing auricular fibrillation; ventricular rate 70 (patient thoroughly digitalized).

An electrocardiographic tracing made on July 30, 1927, showed auricular fibrillation with a ventricular rate of 70, inverted T wave in second and third leads and QRS interval of 0.06 seconds.

The administration of quinidine sulphate was begun on Aug. 10, 1927, starting with a small dose to test for sensitivity; this was followed by gradually increasing amounts. On August 15, the patient complained of nausea and weakness after receiving four doses of 6 grains (0.4 Gm.) each at intervals of three hours. Ringing of the ears or vertigo was not complained of, but it was thought advisable temporarily to discontinue the drug. On August 18, six doses of 5 grains (0.324 Gm.) each were again given and the following morning the patient's arrhythmia had disappeared. Up to this time he had received 119½ grains

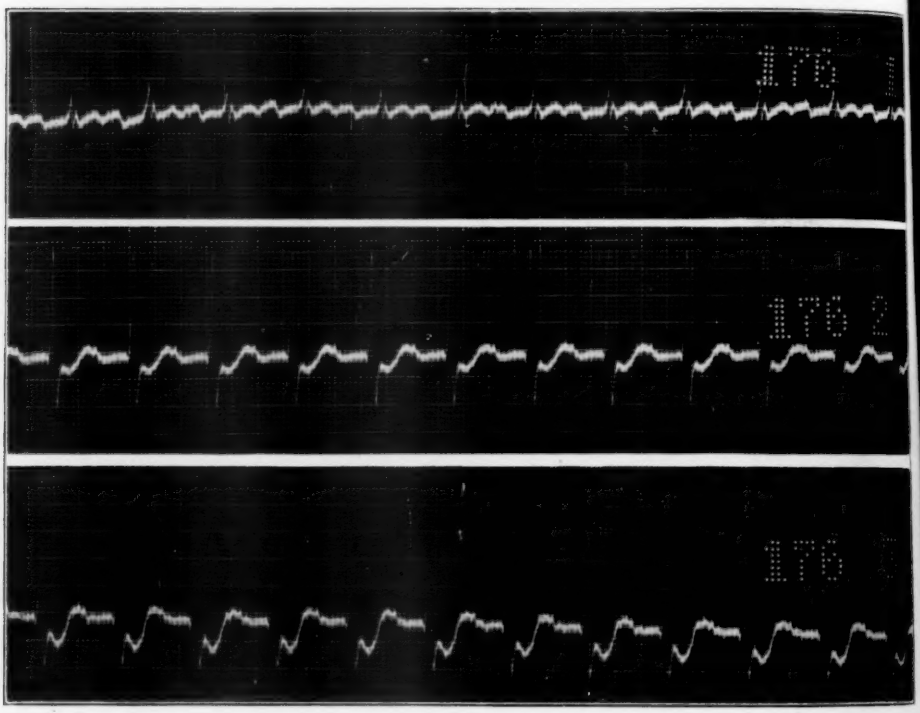

Fig. 3.—Electrocardiogram of J. L., showing restored sinus rhythm after administration of quinidine sulphate. A-V interval prolonged to 0.21 seconds as result of impaired conduction mechanism.

(7.77 Gm.) of quinidine. An electrocardiographic tracing made Aug. 20, 1927, showed restoration of sinus rhythm with a rate of from 90 to 100 and an A-V interval prolonged to 0.21 seconds.

The patient is now on a maintenance ration of 5 grains of quinidine sulphate daily.

CASE 2.—Elizabetr I., aged 11½ years, was admitted to the Seaside Hospital on May 25, 1927, with a diagnosis of chronic rheumatic heart disease. Her tonsils had been removed at the age of 6 because of recurring sore throat. She had had several attacks of rheumatic fever since then. She spent a month at the Seaside

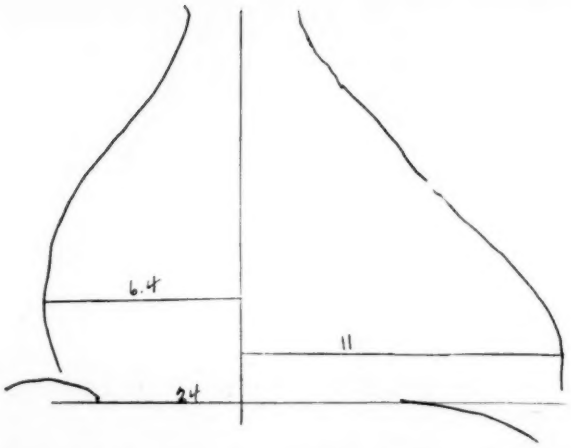

Fig. 4.—Film of the heart at six feet shows an extreme diffuse enlargement of the heart shadow. The enlargement is fairly symmetrical. The picture illustrates the degree of cardiac decompensation.

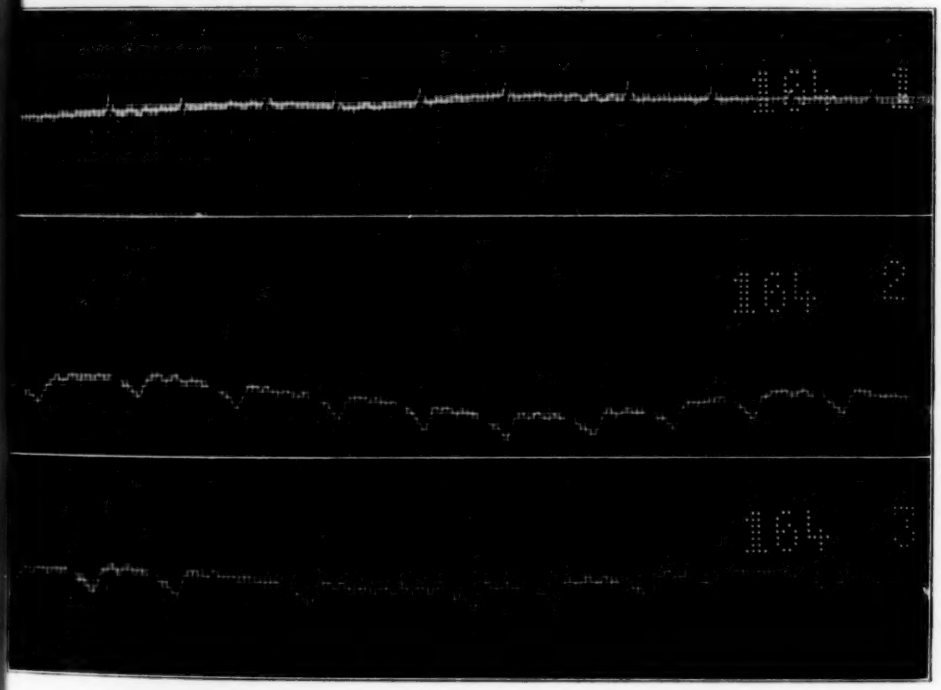

Fig. 5.—Electrocardiogram of E. I., showing auricular fibrillation. The high R-wave in lead III suggests right preponderance.

Hospital in the summer of 1926. At this time cardiac enlargement with systolic and diastolic murmurs was noted, and a diagnosis of mitral stenosis and insufficiency was made. A film of the heart taken at 6 feet showed a marked and fairly uniform enlargement of all chambers. The transverse diameter of the heart was 15.7 cm. and the internal diameter of the chest, 24.5 cm. Heart action was regular except for some sinus arrhythmia. She received heliotherapy and graded exercises and was discharged improved. She went to school until February, 1927. From then until her present admission at the hospital, she had been at home in bed most of the time.

Physical examination on admission revealed a large, well developed and nourished, but pale and slightly cyanotic child. She was nervous and had vomited all of her first meal at the hospital. The heart was markedly enlarged to palpation and percussion. The pulse rate was 156 at the wrist (through an oversight the apex rate on admission was not recorded), the rhythm totally irregular, and the beats extremely variable in intensity. The pulmonic second sound was accentuated. There was a blowing systolic murmur heard all over the precordium, but loudest at the apex. The tonsils had been removed, but considerable tissue remained in one fossa. The liver was palpable two finger-breadths below the costal margin, but pulsation was not made out. The lungs were clear. There was no ascites or edema.

A film of the heart taken at 6 feet showed an outline similar to that noted the preceding summer, with the dilatation more marked. The total transverse diameter was now 17.4 cm., an increase of 1.7 cm. in a year.

The patient was given 20 minims (1.25 cc.) of tincture of digitalis three times daily, and the pulse rate rapidly fell to about 100. The pulse deficit persisted for some time and tended to recur, but completely disappeared when the heart rate was kept at 80 or below. An electrocardiographic tracing made July 30, 1927, revealed auricular fibrillation with right preponderance The patient had an intermittent slight fever up to 100 and 101 F. for many weeks. A month after entry, she was allowed to be up in a chair. Some walking was permitted a few days before discharge and was well borne. The patient went home considerably improved, Aug. 24, 1927.

The use of quinidine in this case was thought inadvisable in view of the extensive myocardial damage as evidenced by extreme cardiac hypertrophy and dilatation and because of lack of knowledge of the duration of fibrillation.

SUMMARY AND CONCLUSIONS

Another instance of auricular fibrillation in a child under 10 years of age is added to the five already on record. Auricular fibrillation is also described in a girl 11½ years old. This condition, although uncommon, must be considered in interpreting the cardiac irregularities of childhood. Its etiology in early life is usually rheumatic heart disease. Diagnosis and therapy present essentially the same problem as in adults.

CARDIOSPASM IN INFANCY AND IN CHILDHOOD

REPORT OF A CASE IN A CHILD ELEVEN YEARS OF AGE *

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Cardiospasm in infancy and childhood is extremely rare. This is evidenced by the fact that in over 20,000 roentgen-ray studies of the gastro-intestinal tract made at Bellevue Hospital over a period of six years, the case reported in this paper represents the first one encountered in a child 11 years of age.

A survey of the literature from 1879 to the present time reveals reports of only eleven authentic cases.

CASES REPORTED IN THE LITERATURE

CASE 1 (Gottstein[1]).—A boy, aged 4 years, complained of dysphagia and vomiting even when small amounts of food were eaten. A sound met with resistance at the cardia. Esophagoscopy showed a normal but contracted cardia, so that the instrument could not be passed. Narcosis failed to relieve this spasm.

CASE 2 (Freund[2]).—A boy, aged 2 years and 3 months, was breast fed until he was 4 months old; then he was weaned and bottle fed. A milk mixture was given, but the child developed a chronic form of vomiting which resisted all medical treatment. Physical examination showed an emaciated, irritable and spoiled child. An attempt to pass a sound met with an obstruction at the cardia. Vomiting ceased immediately after this procedure and did not recur.

CASE 3 (Méry[3]).—A girl, aged 10 months, was breast fed for three months. In the fourth month, she had an attack of asphyxia; from this time on, she gained little in weight. Attacks of vomiting accompanied by slight laryngeal stridor and some hiccup occurred immediately after the ingestion of fluids on three occasions. Méry[3] gave the infant milk of bismuth. Roentgen-ray examination revealed a black mass above the stomach. In about thirty seconds, part of the mass was seen to enter the stomach while the remainder was ejected.

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* Received for publication, Nov. 26, 1927.

* From the Department of Diseases of Children, College of Physicians and Surgeons, Columbia University, and the Children's Medical Division and the Department of Radiography, Bellevue Hospital.

1. Gottstein, G., quoted by Freund: Monatschr. f. Kinderh. **2**:15 (Aug.) 1903.
2. Freund, W.: Kardiospasmus, vermütlich hysterischer Natur bei einem 2 jährigen Kinde, Monatschr. f. Kinderh. **2**:15 (Aug.) 1903.
3. Méry, quoted by Adams: Arch. Pediat. **26**:170 (Feb.) 1909.

Report of Eleven Authentic Cases of Cardiospasm in Children from 1879 to the Present Time

No.	Author	Sex*	Age When First Seen	Age of Onset	Duration	Chief Complaint	Emaciation	Sounding	Roentgen-Ray or Fluoroscopic Examination	Cause of the Spasm	Treatment	Result
1	Gottstein...	♂	4 yr.	?	?	Dysphagia and vomiting	Yes	Obstruction at the cardia	Not done	Unknown	?	?
2	Freund.....	♂	2½ yr.	4 mo.	2 yr.	Vomiting	Yes	Obstruction at the cardia	Not done	Hysteria	Passage of a sound and "suggestive" treatment	Cured
3	Méry.....	♀	10 mo.	4 mo.	?	Vomiting	Yes	Not done	Spasm of the cardia which relaxed in 30 seconds	Swallowing of air	?	?
4	Adams.....	♀	6 mo.	4½ mo.	Died	Dysphagia and vomiting	No	Obstruction at the cardia	Not done	Pressure of tracheobronchial gland	Feeding through a catheter introduced into the stomach	Died of bronchopneumonia
5	Caillé.....	♂	10 yr.	?	?	Vomiting	No	Not done	Spasm of the cardia	Hydrocephalus (?)	Feeding through a stomach tube	Improved
6	Göppert....	♂	8 mo.	4 mo.	4 mo.	Vomiting	Yes	Obstruction at the cardia	Not done	Unknown	Passage of a sound	Cured
7	Beck.....	♂	3 mo.	?	?	Vomiting	Yes	Obstruction at the cardia	Not done	Unknown	Nutrient enemas	Cured
8	Beck.....	♂	2 mo.	?	?	Regurgitation resembling rumination	No	Obstruction at the cardia	Not done	Unknown	Nutrient enemas	Cured
9	Bruce.....	♂	3 yr.	Since birth	3 yr.	Choking, pain over xyphoid and vomiting	Yes	Not done	Spasm of cardia	Neurosis	Atropine sulphate and benzyl benzoate	Cured
10	Castronovo	♂	5 yr.	Since early life	?	Regurgitation, choking	No	Not done	Fusiform dilatation of esophagus with spasm of cardia; Emptied after 25 minutes	Congenital	?	?
11	Seeger.....	♂	12 yr.	3 yr.	9 yr.	Dysphagia, vomiting, pain in the chest	Yes	Obstruction at the cardia	Obstruction at the lower end of esophagus; moderate dilatation of the esophagus	?	One treatment with Plummer's hydrostatic dilator	Cured

* In this table ♂ indicates male, ♀ female.

Case 4 (Adams[4]).—A girl, first seen when 6 months of age, was nursed for the first week after birth and then given a formula. The infant did poorly and was constantly troubled with a "sour stomach." At the age of 4½ months, she began to have difficulty in swallowing, and she would either vomit soon after each feeding or the food would be retained for several days and then the vomiting would recur. The vomitus did not come from the stomach. A few teaspoonfuls of water were given, which were vomited after several minutes. An ordinary sized feeding tube met with an obstruction just above the stomach. A number 10 French catheter passed the obstruction. After this, the attacks occurred every two weeks or so and lasted for from one to two hours. The patient finally died of an intercurrent bronchopneumonia. Postmortem examination of the esophagus showed a marked circular narrowing 5 inches above the cardia. The narrowing was due to a markedly uniform induration between the walls. Immediately adjacent to the induration on the left side a bronchial gland the size of a chestnut was found, which was pressing inward on the tube. A well defined left-sided diverticulum about the size of a number 10 bird shot was noted immediately above the stenosis. Just below the stenosis on the anterior wall, two other diverticula were noted; they were about the same size as the one described. The gland was tuberculous.

Case 5 (Caillé[5]).—A boy, aged 10 years, complained of vomiting. Roentgen-ray examination showed a spastic condition of the cardia but no diverticula. The patient was fed through a tube which had been introduced into the stomach. Improvement was not complete. It was Caillé's opinion that the spasm might have been of central origin, since the boy had had hydrocephalus in infancy.

Case 6 (Göppert[6]).—A boy, aged 8 months, was breast fed until he was 3 months old and made satisfactory progress. During the fourth month, zwiebach was added to his diet. At this time, he developed intractable vomiting, which occurred from ten to thirty minutes after meals. The vomitus did not contain acid and did not show digestive changes. A 5 mm. sound met with obstruction at the cardia; a 4 mm. sound, however, could be introduced into the stomach. Through a catheter passed nasally into the stomach, the infant was given 120 Gm. of milk. This milk was retained, which showed that the cause of the obstruction was in the cardia. For four days, feedings were retained, and then the vomiting recurred. Sounds were passed again and met with an obstruction at the same place. Improvement followed the introduction of these sounds, and the feedings were retained.

Case 7 (Beck[7]).—A boy, aged 3 months, was breast fed for eight days and then bottle fed. Soon after the change in feeding he began to vomit. He was admitted to the hospital and placed on a diet of malt soup mixture. Vomiting continued, but only at rare intervals. One month after admission he suddenly began to vomit after each feeding. The vomitus did not show digestive change. A sound met with an obstruction at the cardia but could be easily pushed past the obstruction into the stomach. Food could be retained only by catheter feeding. Nutrient enemas were now instituted and were well tolerated. In a few days, fluids were readily retained, and in two weeks the infant was completely cured.

4. Adams, S. S.: A Case of Cardiospasm, Arch. Pediat. **26**:170 (Feb.) 1909.

5. Caillé, in discussion on Adams (footnote 4).

6. Göppert, F.: Der Kardiospasm, eine Ursache des habituellen Erbrechen der Säuglinge, Therap. Monatschr. **22**:390, 1909.

7. Beck, C.: Kardiospasmus in Säuglingsalter, Monatschr. f. Kinderh. **9**:155, 1910.

Case 8 (Beck[7]).—A boy, aged 2 months, was admitted to the hospital for intractable vomiting and was put on a diet of malt soup mixture. He would choke on each swallow, regurgitate and then reswallow the food. This was repeated at each feeding. An attempt to pass a 5 mm. sound into the stomach met with an obstruction at the cardia. Feeding through a catheter which had been introduced into the stomach did not result in vomiting. Various formulas to stop the vomiting were tried, but all failed. Nutrient enemas were now given and were tolerated. In a few days, fluids in small amounts could be retained, and in a few weeks the infant was cured.

Case 9 (Bruce[8]).—A boy, aged 3 years, had had attacks of choking when fed since birth. The first attack occurred a few hours after birth, and for one year he had had one or more attacks each day. Since that time, the attacks had been less frequent. When seen at 3 years of age, the attacks occurred once or twice a month. Choking would begin after a few mouthfuls of food had been swallowed. The child would then complain of pain in the xiphoid region. This was followed by a cough, which became more and more violent, and which subsided only after vomiting had occurred. The physical examination showed an undernourished, pale, weak child with a distended tympanitic abdomen. A diagnosis of chronic intestinal indigestion was made, and treatment for this was instituted. The patient was seen while he had one of these attacks and it was noted that the vomitus was unchanged milk, and that it was not sour. It was therefore assumed that the trouble was in the esophagus and not in the stomach. Fluoroscopy revealed a spasm of the cardia. Treatment consisted in giving atropine to physiologic limits and also benzyl benzoate. After three doses the child was able to retain a glass of milk. The spasm did not return.

Case 10 (Castronovo[9]).—A boy, aged 5 years, when first seen, had a history of vomiting and regurgitation since birth. Sometimes the patient would vomit the food eaten twelve hours before. The regurgitation resembled the act of rumination. The frequency of the attacks varied. He never complained of pain. Physical examination showed a healthy child. Roentgen-ray examination of the gastro-intestinal system revealed a normal stomach, duodenum and pylorus. There was a fusiform dilatation of the esophagus, the lower end of which projected in the shape of a cone; and the tip of the cone was at the cardia. After a few minutes, a contraction of the most dilated part of the esophagus could be seen, and the level of the food rose into the pharynx and fell again with the relaxation of the contraction. The cardia remained tightly shut. In a few minutes, a strong contraction forced a part of the food into the child's mouth, and this food was reswallowed. Twenty-five minutes later, the cardia relaxed, and the esophagus was empty.

Case 11 (Seeger[10]).—A boy, aged 12 years, complained of dysphagia, regurgitation, pain in the chest and occasional difficulty in breathing. There was no difference in his ability to swallow water or solids. Regurgitation usually followed soon after eating, but occasionally he would not vomit until several hours after the food had been taken. Roentgen-ray examination showed a definite and sharp

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8. Bruce, J. W.: A Case of Cardiospasm in a Three Year Old Child, Kentucky M. J. **19**:824 (Dec.) 1921.

9. Castronovo, E.: Congenital Spasm of the Cardia in a Child Six Years of Age, Pediatria **33**:483 (May) 1925.

10. Seeger, S. J.: Cardiospasm: A Report of a Case in a Boy of Twelve, Wisconsin M. J. **26**:154 (March) 1927.

line of obstruction at the cardia with moderate dilatation of the esophagus. After four hours, the esophagus was empty. A number 43 olive bougie was passed, and it encountered some obstruction at the cardia. Treatment consisted in using Plummer's hydrostatic dilator. Immediately after the first dilatation, the child was able to swallow both liquids and solids.

REPORT OF AUTHORS' CASE

History.—M. C., a girl, was first seen in the outpatient department of the Bellevue Hospital in December, 1926; she was then 10 years and 5 months of age. The chief complaint was pain in the chest of two months' duration. The family history was unimportant. Delivery occurred at full term and was normal. Postnatal and developmental history were normal. She had had measles at the age

Fig. 1.—Appearance of the patient two weeks after admission to the hospital.

of 6 and chickenpox at the age of 7. Her appetite was good; movement of the bowels was regular, and digestion was normal.

Examination.—Physical examination showed an anemic, undernourished child, 48 inches (121.9 cm.) in height, weighing 43 pounds (19.9 Kg.); the average weight for her height was 53 pounds (24.0 Kg.). The skin was dry, rough and warm. Her posture was poor. She had a left dorsal scoliosis and a moderate lumbar lordosis. The lower part of her abdomen was sagging. A diagnosis of malnutrition was made, and appropriate treatment was instituted.

Treatment and Course.—She was next seen on Sept. 6, 1927. Five months prior to the visit she had contracted pertussis. The attacks had been typical of the disease. The cough and the vomiting, however, though diminished, had persisted. On close questioning, it was found that she had been exposed to an active case of pulmonary tuberculosis in a maternal cousin. The child was still markedly undernourished and weighed 43 pounds (19.9 Kg.), a loss of 3 pounds (1.4 Kg.) in the past seven months. The lungs were normal, except for the

presence of D'Espine's sign to the sixth dorsal vertebra. Our first impression was that the vomiting and the cough were due to enlarged tracheobronchial glands. When the patient was seen one week later, cough and vomiting were still present. In addition, she complained of a pain in the chest. The results of a physical examination were negative. The oral temperature was 100 F. All of these facts suggested the possibility of an active tuberculosis, and she was therefore admitted to the hospital for further study.

After the first four days in the hospital, the patient had a daily afternoon rise in temperature to 100 F.; thereafter the temperature was normal. The Mantoux test was negative to both 0.05 mg. and 0.2 mg. of old tuberculin. Repeated examinations of the sputum failed to show acid-fast bacilli. Roentgen-ray examinations of the chest did not give evidence of pulmonary disease.

The results of a urinalysis were negative. Blood examination showed: white cells, 8,400; polymorphonuclear leukocytes, 75 per cent; red cells, 3,800,000; hemoglobin (Sahli), 70 per cent. The Wassermann reaction was negative. A chemical examination of the blood was normal. It contained: total nonprotein, 19.1 mg.; uric acid nitrogen, 1.0 mg.; chlorides, 573 mg.; cholesterol, 174 mg.; phosphorus, 2.6 mg. per hundred cubic centimeters of blood; plasma protein, 7.95 Gm.; hemoglobin content, 64 per cent, and cell volume, 34 per cent.

A few days after admission, the cough ceased. The vomiting, however, persisted and occurred about once or twice a day, at times immediately after the ingestion of food and at other times several hours after a meal. She had no difficulty in swallowing and did not complain of pain.

As tuberculosis could be excluded on the basis of the negative observations, a lesion of the gastro-intestinal tract was suspected as being the primary factor in causing the vomiting. She was given a barium meal, and six hours later, a roentgen-ray examination revealed the presence of a spasm at the cardiac end of the esophagus. Half the barium meal was still within the esophagus, which was dilated to twice its normal size. There were no organic lesions of the stomach or of the duodenum. The vomitus was now examined, and it was found to consist of undigested food.

The cause of the intractable vomiting was now evident. Enough tincture of belladonna was given to produce a physiologic effect, but it did not have any influence on the vomiting. Four days later, a fluoroscopic examination still showed the presence of marked cardiospasm. Only a little of the barium was noted entering the stomach. The patient was now given a hypodermic injection of atropine sulphate, 1/150 grain (0.003 Gm.). Fifteen minutes later, the esophagus was seen practically to empty itself. Three days later, a fluoroscopic examination was made again. Examination six hours after the barium meal showed a retention of one-half the barium in the esophagus. Fifteen minutes after a subcutaneous injection of atropine sulphate, 1/150 grain, there was no change. After another fifteen minutes, the esophagus was empty.

Vomiting still continued at irregular intervals. It was observed that solids and hot foods were retained better than liquids and cold foods. At no time did the patient complain of dysphagia. Her appetite was always excellent. The twenty-four hour output of urine averaged as little as 250 cc., which was probably due to the diminished fluid intake. She was extremely constipated. The vomiting continued irrespective of frequent changes in the diet and of medication, which consisted of atropine, bromides and sodium bicarbonate.

On October 15 (four weeks after her admission to the hospital), esophagoscopy was performed to discover, if possible, a local etiologic factor. The report was: "A 7 mm. esophagoscope was passed. Just above the hiatus there was

considerable thickening and congestion of the mucous membrane lining the esophagus. At the hiatus there was considerable angulation anteriorly and to the left with spasm which made it difficult to pass the esophagoscope into the stomach."

Immediately following the procedure, the child complained of excruciating generalized pain in the chest and in the right epigastrium. Half a grain of

Fig. 2.—Roentgenogram of the esophagus showing cardiospasm and moderate dilatation of the esophagus.

codeine sulphate gave some relief. Twelve hours later, the temperature rose to 103 F., and an examination of the lungs revealed the presence of a friction rub in the right axilla with dulness and suppressed breath sounds over the right lower lobe anteriorly. The temperature continued high, ranging from 102 F. to 105 F. Two days later there was evidence of a massive right pleural effusion and a pleuritic friction rub was heard over the left anterior side of the chest.

The child was given a fluid diet consisting of an alkaline water, milk and orange juice. The blood culture did not show growth after seventy-two hours. Fluid was aspirated from the right side of the chest, and 500 cc. of a turbid, amber-colored, sour-smelling fluid was removed. A direct smear of the fluid showed numerous short-chained streptococci and many diphtheroid bacilli with a few pus cells. As we suspected that we were dealing with an esophagopleural fistula, we administered methylene blue orally, and again performed thoracentesis. The aspirated fluid was streaked with blue. Microscopic examination of the

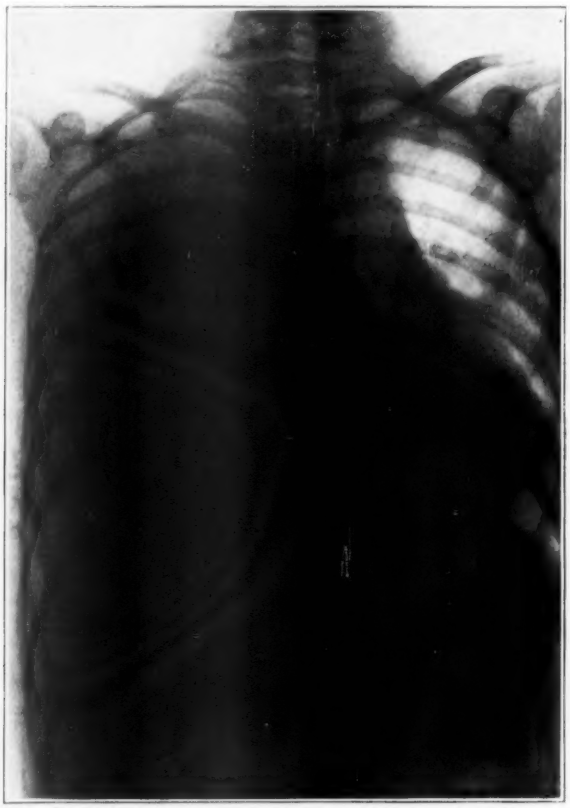

Fig. 3.—Roentgenogram of the chest showing massive right pleural effusion.

fluid showed a marked resemblance to milk. The diagnosis was now evident; food and medication by mouth were therefore discontinued. Nutrient enemas were instituted, and fluids were administered by hypodermoclysis. She became rapidly worse and died six days after esophagoscopy. Postmortem examination was not obtained.

It was later learned that the child had complained of pain in the chest on breathing with occasional cough during sleep for three nights preceding the esophagoscopy. There is a possibility that ulceration with esophagopleural adhesions already existed, which made it easy for perforation to occur with the least force.

COMMENT

Etiology.—Various possible factors have been suggested as the cause of cardiospasm. Bruce [8] believes it to be a functional disturbance of neurotic origin. In some cases, however, it may be due to toxic causes, for example, those following influenza or pneumonia. Held and Gross,[11] in a thorough discussion of the etiology of cardiospasm, state that it is due essentially to hyperirritability of the vagus. They divide the underlying factors into five groups:

(*a*) A defective inherited vegetative nervous system, manifested by signs of vagotonia or of sympatheticotonia or of the two types mixed.

(*b*) A defective vegetative nervous system because of an inborn status asthenicus or status thymicolymphaticus. In this group, the hyperirritability of the vagus or of the sympathetic system is secondary. In this group belong the cases of cardiospasm resulting from sudden psychic trauma.

(*c*) Reflex irritation from other diseased organs, as from diseases of the genito-urinary tract or from left-sided pleurisy, etc.

(*d*) Spasm due to toxic or a metabolic agent.

(*e*) Spasm due to local diseases, such as fissure, erosion and scar tissue formation.

Castronovo [9] divides cardiospasm in infants and children into four groups:

(*a*) Essential spasm without any demonstrable organic changes.

(*b*) Spasm due to a lesion of the esophagus.

(*c*) Spasm of early infancy due to being given food that was not wanted.

(*d*) Spasm that occasionally occurs in patients suffering from congenital pylorospasm and stenosis.

He emphasizes a neuropathic and especially a hysterical taint in the idiopathic cases.

Essentially pure cardiospasm is of neuropathic origin and is due to a functional disturbance of the vagus nerve. In our case, the remarkable effect of atropine in causing a relaxation of the spasm sufficient to permit the contents of the esophagus to empty completely within thirty minutes points to the vagus nerve as the possible underlying cause in this disturbance. The close relationship of the onset of the attack of whooping cough to the cardiospasm may be of significance. It is suggested that whooping cough may have been a contributing factor in the production of the cardiospasm, because of the strikingly close relationship to the onset of the esophageal condition.

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11. Held, I. W., and Gross, M. H.: Cardiospasm, J. A. M. A. **66**:233 (Jan. 22) 1916.

Symptoms.—As a rule, the onset is sudden. The chief complaint is vomiting, which usually occurs immediately after the ingestion of food, but food may be retained for several hours and then vomited. Warm foods and solids are retained better than cold foods and liquids. The regurgitated food is never sour and does not show evidence of digestive changes; but in cases of extreme dilatation of the esophagus, the food may show fermentative changes. Dysphagia is an uncommon symptom in children. Pressure symptoms may be present owing to stagnation of the food. The presence of pain, however, is variable and is usually located over the xiphoid region. When the spasm is sufficiently extreme to force some of the contents into the pharynx and larynx, coughing results. The character of the act of regurgitation at times shows a striking resemblance to rumination, with which condition it has probably been confused. Loss of weight and emaciation are outstanding symptoms in many cases. Owing to the low fluid intake, constipation, oliguria and dryness of the skin occur.

Confirmatory evidence of obstruction at the cardia is the delay or absence of the normal gurgling sound heard at the lower end of the sternum or posteriorly at the level of the tenth dorsal vertebra to the left of the median line. Further proof may be obtained by passing a bougie or a sound which meets with an obstruction at the cardia in cases of spasm. Esophagoscopy is also helpful, not only in demonstrating the spasm, but in clearly differentiating this condition from actual organic disease. Rosenheim [12] and Stark [13] observed hyperemia in some cases. In others, there was an ischemia above the spasm and also some folds enclosing a narrow, half-moon slit or even a pin-point opening. Our patient showed considerable thickening and congestion of the mucous membrane just above the hiatus.

The clearest and most convenient means of determining the presence of cardiospasm is by a roentgen-ray examination. This shows a smooth contour of the entire esophagus which tapers to a point at its lower end; very little, if any, of the barium enters the stomach. The dilatation above the spasm is of a variable degree.

Differential Diagnosis.—Cardiospasm in infants and in children must be differentiated from several conditions. It is distinguished from pylorospasm and stenosis by the unchanged character of the vomitus, by the absence of visible peristaltic waves of the stomach and by the absence of a palpable tumor. In rumination, the vomitus consists of digested food. In cases of stricture, there is a history of trauma or of the swallowing of caustics. Roentgen-ray examination will definitely differentiate the two conditions. An esophageal diverticulum can best be excluded by the roentgen ray.

12. Rosenheim, quoted by Held and Gross (footnote 11).

13. Stark, quoted by Held and Gross (footnote 11).

Treatment.—The most effective form of treatment is dilatation of the spasm by means of bougies or by means of the Plummer apparatus. The results, however, are frequently not permanent. Vinson [14] reports that 25 per cent recur within the first six months. Atropine is of value, but its effect is transient. Bromides may be of value in some cases.

SUMMARY

1. The literature on cardiospasm in infancy and in childhood is reviewed, and eleven authentic cases are tabulated.

2. An additional case is reported in a girl, 11 years old, which followed closely an attack of whooping cough; roentgen-ray study showed a large six hour esophageal retention.

3. The etiology, symptoms and treatment in this condition are discussed.

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14. Vinson, P. P.: Diagnosis and Treatment of Cardiospasm, J. A. M. A. **82**:859 (March 11) 1924.

PERFORATED GANGRENOUS MECKEL'S DIVERTICULUM IN A NEW-BORN INFANT

REPORT OF A CASE *

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The persistence of remains of the embryonic omphalomesenteric duct in the human being is by no means rare. The statistics compiled by Griffith [1] show an incidence of from 1 to 2 per cent. Christopher [2] gives the percentage as approximately 2 per cent, as found in autopsy statistics. The incidence in surgical practice varies considerably. In a study of 10,600 laparotomies at the Mayo Clinic, Balfour [3] found only fifteen instances of Meckel's diverticulum (0.14 per cent). In abdominal operations Coleman [4] has made a routine examination of the lower ileum whenever possible; in approximately 1,000 operations, he has found Meckel's diverticulum eighteen times (1.8 per cent). Nearly all writers have emphasized the predominance of the structure in males, the ratio being nearly 3 to 1.

The age at which Meckel's diverticulum may give rise to symptoms is of interest. Halstead [5] states that obstruction from a diverticulum usually occurs in children or young adults. Porter,[6] in a study of 137 cases in which the age was given, found the average to be 21 years. In Wellington's [7] series of 326 cases, the age ranged from 5 months to 60 years. The literature contains numerous reports of complications arising from the presence of omphalomesenteric remains in young children, but in the new-born child this appears to be rare. For this reason, it may be of interest to pediatricians to record the observations in a case of this kind which I have recently had the opportunity to study.

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* Received for publication, Sept. 20, 1927.

* From the Department of Pathology, University of Oregon Medical School.

1. Griffith, J. P. Crozer: Diseases Connected with Meckel's Diverticulum, Tr. Am. Pediat. Soc. **26**:211, 1914.

2. Christopher, Frederick: The Surgical Diseases of Meckel's Diverticulum, Internat. Clin. **1**:67 (March) 1925.

3. Balfour, D.: Meckel's Diverticulum: A Report of Fifteen Cases, J. Minn. State M. Assn. **31**:110, 1911.

4. Coleman, R. C.: Incidence of Meckel's Diverticulum, J. Iowa State M. Soc. **16**:238 (May) 1926.

5. Halstead, A. E.: Intestinal Obstruction from Meckel's Diverticulum, Ann. Surg. **35**:471, 1902.

6. Porter, M. F.: Abdominal Crises Caused by Meckel's Diverticulum, J. A. M. A. **45**:883 (Sept. 23) 1905.

7. Wellington, J. R.: Meckel's Diverticulum, Surg. Gynec. Obst. **16**:74, 1913.

Cawardine [8] has described an instance of volvulus of a Meckel's diverticulum in a child 2 days old. Gluck [9] found that in a child born at 7 months with an umbilical hernia in whom signs of peritonitis and strangulation developed, the symptoms were caused by a mesenteric fold bound to a Meckel's diverticulum and passing over the intestine. Hallet [10] saw a girl 2 hours old, with an irreducible umbilical hernia containing the cecum and small intestine, with an adherent Meckel's diverticulum. Briddon [11] has reported a somewhat similar case in a child 10 days of age. A recent report is that of Mathews [12] describing an instance of umbilical hernia with an attached diverticulum occurring in a boy only 20 hours old.

A fairly thorough search of the literature has revealed only one case similar to the one here reported. Shukowski [13] saw an infant who was normal until the third day of life, when it refused to take nourishment and began to vomit frequently. Early in the day there was a diarrhea, followed by a constipation. The temperature was subnormal, being from 30 C. (86 F.) to 35.2 C. (94.8 F.). Examination of the abdomen revealed marked distention; the upper half was soft and the lower portion firm; the skin was edematous; the liver was not palpable, and the umbilicus protruded slightly but was otherwise normal.

The vomiting and constipation continued. On the fourth day, symptoms of collapse appeared; the extremities and face became cold; the skin was pale and the heart action weak. An enema given with the hope of relieving the abdominal distention was followed almost immediately by death.

Autopsy revealed a large quantity of free gas in the peritoneal cavity. This had forced the liver and transverse colon upward. In the lower half of the abdomen the small bowel, cecum and sigmoid were matted together by fibrin, and on the surface of the bowel, mucus and bits of meconium were recognizable. The colon and rectum were empty. At the junction of the jejunum and ileum and on the mesenteric side was an opening the size of a pea. From the appearance of the perforation and its location, Shukowski believed that there had existed a Meckel's diverticulum which had perforated and given rise to the peritonitis. The kidneys contained many uric acid infarcts. The lungs and heart were normal.

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8. Cawardine, T.: Brit. M. J. (Dec. 4) 1897, p. 1637.

9. Gluck: Berl. klin. Wchnschr. (Jan. 19) 1885; cited by Bunts, F. E.: Ann. Surg. **40:** 1904, p. 536.

10. Hallet: Rev. de Gynec. et de chir. abd. (June, 1900); cited by Bunts (footnote 9).

11. Briddon: Ann. Surg. **27:**647, 1888; cited by Porter (footnote 6).

12. Mathews, F. S.: Meckel's Diverticulum: Report of Twelve Cases Including One Tumor, Arch. Surg. **10:**720 (March) 1925.

13. Shukowski, V. P.: Perforated Peritonitis in New-Born, Med. Obozr. Mosk. **57:**117, 1902.

REPORT OF A CASE

The infant was a patient of Dr. Harry B. Moore, to whom I am indebted for the following clinical data.

History.—Baby B., a boy, was born at 7:20 a. m. on May 6, 1927. His weight at birth was 6 pounds and 9 ounces (2.95 Kg.). The mother was a primipara, aged 34. Her pregnancy was normal until the seventh month, at which time a trace of albumin appeared in the urine and the blood pressure increased from 130 to 168 (systolic). On this account labor was induced at the end of eight and one-half months. Labor and delivery were normal.

On the second and third days of life, the baby took the formula and breast. A slight rise in temperature (99.2 F.) on the third day was thought to be due to inanition. The bowel and renal excretions were normal.

On the fourth day, the temperature rose, first to 100.4 F. and later to 101 F. The weight was now 6 pounds (2.7 Kg.). Glucose administered by rectum was not retained and milk by mouth was vomited. At 5 p. m. on this day, the baby was seen in consultation with Dr. W. F. Patrick, whose observations were as follows: Moderate cyanosis of the skin of the face and abdomen, dyspneic breathing, and increased heart rate. Examination of the abdomen disclosed edema of the wall, marked distention and boardlike rigidity; evidence of infection was not found about the navel. The clinical diagnosis was: an acute condition of the abdomen of undetermined cause, which required surgical attention.

During the evening the vomiting continued, and the infant died at 11 p. m.

Autopsy Observations.—Autopsy was performed on May 10, 1927. The body was that of a male child 4 days of age and weighed about 6 pounds (2.7 Kg.). External evidence of injury or disease was not seen. The fontanels were normal in size; the head was well formed; there was no evidence of a caput succedaneum. About the nostrils, lips and mouth were yellowish-brown stains. The thorax was normal. The abdomen was greatly distended and rounded; the cord was dry and did not show any trace of infection. The skin was livid everywhere.

A considerable quantity of gas escaped when the abdominal cavity was opened. The umbilical vein, hypogastric arteries and urachus were closed. In the peritoneal cavity was a moderate amount of yellowish fluid which contained fibrin and whitish curds of milk. The parietal peritoneum was reddish and coated with fibrin. The urinary bladder was empty.

The thymus was of average normal size. The pericardial sac was free from changes; the right chambers of the heart appeared somewhat dilated; the foramen ovale was patent, and a postmortem clot extended through it into both auricles; the ductus arteriosus was likewise anatomically patent; the myocardium appeared pale and pasty. The mucosa of the esophagus was dark from bile staining. The lungs were fairly well aerated and did not exhibit evidence of consolidation.

The inner cortical zones of the suprarenals were dark, the outer zone pale. The kidneys showed some degree of fetal lobation, and on section the pyramids stood out prominently, owing to the presence of numerous uric acid infarcts. A pea-sized supernumerary spleen was located in the gastrolienic ligament; the only change in the spleen proper was congestion. The stomach contained bile-stained fluid; the duodenum was normal. The liver was of average normal size, its capsule was partly covered with fibrin, and the cut surfaces showed cloudy swelling. The pancreas was normal.

In the ileum, 20.4 cm. from the ileocecal junction, was a Meckel's diverticulum (fig. 1), the visible portion of which lay over the anterior surface of the ileum

with the distal end directed slightly proximally along the bowel. The structure resembled a nursing nipple, having a stem and a bulbous end.

The total length of the visible free portion of the diverticulum was 2.2 cm., with a diameter at the base of 0.6 cm. and of 1.1 cm. at the distal enlargement. In the distal end was a ragged opening, the margins of which were surrounded by a fibrinopurulent exudate. Food material from the bowel could be expressed from this opening. The entire bulbous portion was dark reddish black and gangrenous. On the proximal side (i. e., toward the jejunum) was a triangular bit of mesentery in which two vessels were visible. The larger of these passed into the wall of the diverticulum at the base of the gangrenous portion. On the anterior surface of the ileum distal to the visible part of the diverticulum was a shallow groove which extended toward the cecum. On opening the ileum, the internal opening of the diverticulum was found to be nearer the cecum than the free portion indicated. The lumen readily admitted a 2 mm. probe. When

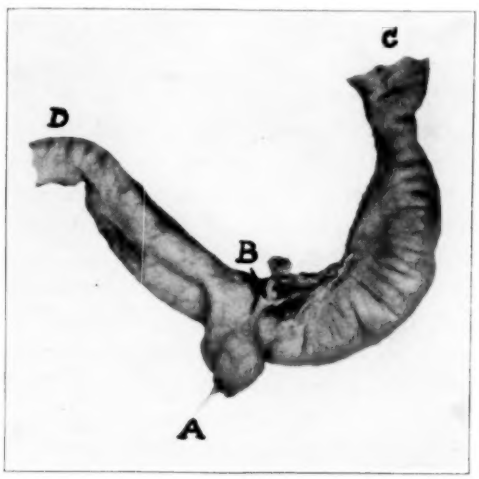

Fig. 1.—Meckel's diverticulum and portion of the ileum (slightly reduced). *A* is a glass marker in the perforation; *B*, an artery supplying the gangrenous end of the diverticulum; *C*, proximal end of ileum; *D*, distal cut end of ileum.

the probe was passed into the opening, it was found that the lumen passed diagonally through the wall of the ileum and above the groove seen externally. The total length of the lumen was 3.8 cm.

Microscopic Observations.—Sections from the mesentery of the diverticulum contained one large and several small arterioles as well as a number of small veins. The arterioles were, without exception, free from blood; the veins, on the other hand, were engorged. The only abnormality found in the arterial walls was postmortem desquamation of endothelium.

Longitudinally cut sections of the diverticulum revealed a mucosa which was identical with that of the small intestine. For about one-half the length of the diverticulum there was a well defined inner circular and an outer longitudinal layer of muscle, but distally there was a fusion of the two muscle layers with a gradual thinning out until it was altogether lacking at the distal end. At the junction of the stem with the bulbous end, there was a sharp transition from

living cells to complete necrosis. Here there was marked edema but only a slight infiltration by wandering cells, which strongly suggested that the underlying cause of the gangrene was circulatory rather than infectious. Over the serosa about the margins of the perforation there was a fibrinopurulent exudate. In the gangrenous area, the veins were congested but the arterioles were empty.

Microscopic sections of the liver, heart and kidneys revealed a moderate degree of parenchymatous degeneration. There was some desquamation of tubular epithelium and congestion of the glomerular vessels in the kidneys. The lungs did not show evidence of pneumonia.

Anatomic Diagnosis.—The anatomic diagnosis was: Meckel's diverticulum with gangrene, perforation, and generalized serofibrinopurulent peritonitis; free gas in the peritoneal cavity; cloudy swelling of liver, heart and kidneys with numerous uric acid infarcts in the latter; general cyanosis of the skin; bile staining of esophageal mucosa, nares and lips; anatomically patent foramen ovale and ductus arteriosus; small supernumerary spleen and normal involutionary softening of suprarenal cortex.

COMMENT

The cause of the gangrene, which in this case clearly preceded and led to the perforation and peritonitis, cannot be established with certainty. Thrombosis or obliterative endarteritis in the artery supplying the gangrenous portion was not present. Microscopically, the appearance of the lesion was that of an infarction rather than of necrosis due to infection. In the absence of any change in the wall of the artery or its lumen, it is possible that there may have been enough torsion on the mesentery and artery, perhaps brought on by peristaltic movements of the bowel, to cut off the blood supply and result in gangrene.

Clinically, the diagnosis of a perforated Meckel's diverticulum in a new-born infant is most difficult, the symptoms being those of an acute surgical condition of the abdomen without localizing signs.

Progress in Pediatrics

THE FATE AND DEVELOPMENT OF THE IMMATURE AND OF THE PREMATURE CHILD

A CLINICAL STUDY

Review of the Literature and Study of Cerebral Hemorrhage in the New-Born Infant *

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PHILADELPHIA

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PHYSICAL DEVELOPMENT OF THE IMMATURE CHILD

About 55 per cent of immature infants live until the end of the first year; 52 per cent reach the school age, and 51 per cent reach the age of puberty; in other words, the mortality after the school age is not any higher than for mature children. This is not true, unfortunately, of the physical as well as of the mental development of the immature infant.

Ylppö, in his calculations, always referred to the conception age, i. e., to the intra-uterine and extra-uterine age of the child. He calculated the intra-uterine age from a fetal weight and length curve, so that when he speaks of a child 5 years of age, and that child was born at the end of seven months of pregnancy, the child's extra-uterine or citizen age is really 4 years and 5 months. I prefer to make my calculations on the basis of the extra-uterine age alone, because when a comparative study is made of the data with measurements of normal children, the latter are also based on the extra-uterine or official age alone.

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Height.—The immature infant has a slender body during the first three to six months, i. e., it is long and thin. After this period a change occurs; fat is deposited, the abdomen becomes larger, and the body gradually assumes an entirely different aspect. There seems to be a lagging in the growth of the extremities, so that one sees a thick long body with relatively short extremities. In children, even normally, relatively shorter extremities are found in comparison to the trunk than in adults, but this difference is still more striking in the immature infant. The baby thus looks shrunken or like a compact miniature edition. Rosenstern said that this condition persists until the third year, when proportionate growth of the trunk and extremities is reestablished. A careful analysis of the curves of the growth of the immature infant tells a different story.

Fig. 1.—*A* shows an immature full term infant with a birth weight of 1,750 Gm. Note the disproportionately short extremities in comparison to the length of the trunk. *B* shows an immature full term infant with a birth weight of 1,620 Gm. Note the mammary enlargement. *C* shows an immature 8 months' infant with a birth weight of 1,970 Gm. Note the megacephalus.

Figures 3 and 4 represent the growth of the body height of immature boys and girls. One sees that the majority of them are below the normal growth line. This subnormality is evident at all ages up to the fifteenth year. It is true that the backwardness in height is more striking during the first six or seven years than during the period from seven to fifteen years. Figure 2 also shows the curve of growth of an average immature infant, and one sees therein that the growth curve of the immature infant is almost parallel to that of the mature infant during the first seven years, except that it runs from 8 to 12 cm. below it. The same fact, namely, that the immature infant remains backward in increase in

height up to the fifteenth year becomes still more striking when a detailed study is made of tables 8, 9, 10 and 11. These results, therefore, differ from those of Ylppö. He maintained that immature infants weighing over 1,500 Gm. remain backward in height only up to 4 years of conception age, and that thereafter they catch up with the mature infants. He said, therefore, that the "zone of injury" for growth in height for immature infants is between only the first and fourth year of life. Forschner-Böke, who reexamined fourteen immature children, finds that at 7 years of age there is still a definite backwardness in increase in height and weight. The figures obtained by my co-workers and me show that the delay in growth persists even past the age of puberty. It is true that a large number of our infants had

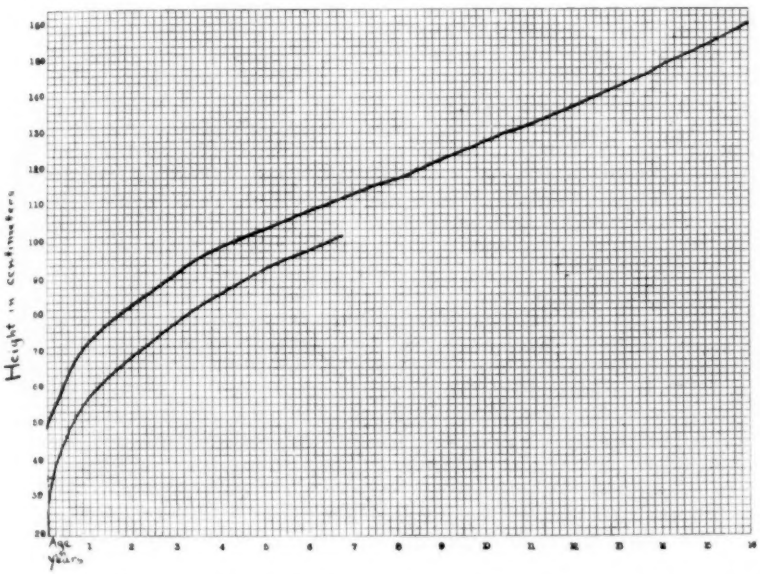

Fig. 2.—Normal growth curve of girls based on Camerer-Pirquet figures, compared with the growth curve of immature infants, portrayed by the shorter curve.

gone through several acute, and some even chronic diseases, and the contention might be brought forward that the delay in growth was due to the influence of these diseases. But clinical experience teaches that children usually increase in height during an illness rather than remain stationary. Furthermore, the greater number of our infants who are most behind as far as increase in height is concerned are those who have not suffered from many acute or any chronic ailments. Comberg also found that of the immature children whom she reexamined, those who had had severe diseases were among those of normal height, and some were even taller than the average mature

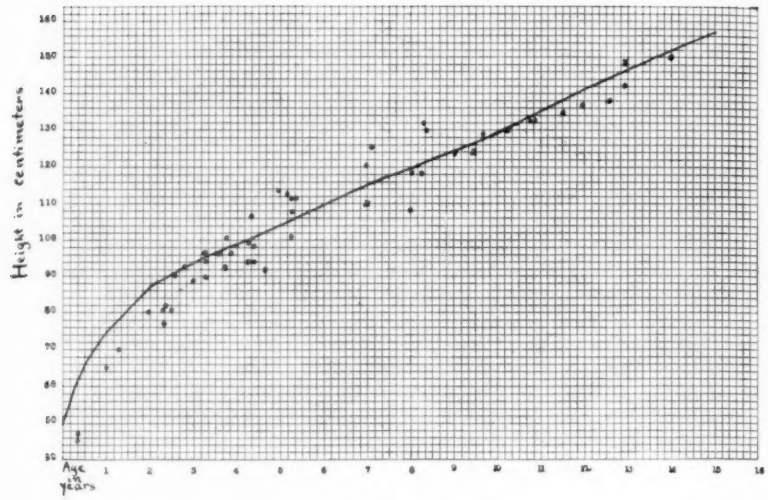

Fig. 3.—Growth curve of immature male infants compared with the normal curve of growth, based on Camerer-Pirquet figures. The crosses indicate infants under 1,500 Gm. of weight; the zeros, infants from 1,501 to 2,500 Gm. of weight.

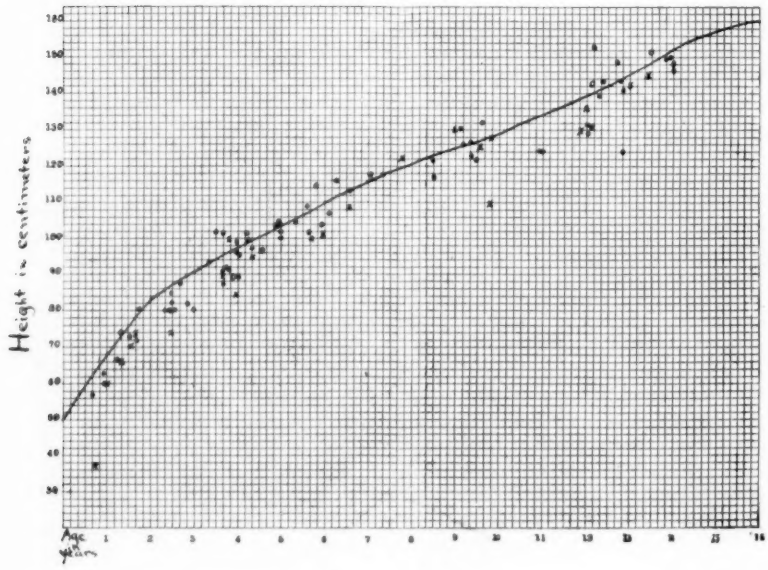

Fig. 4.—Growth curve of immature female infants in comparison with normal growth curve, based on Camerer-Pirquet figures. The crosses indicate infants under 1,500 Gm. of weight; the zeros, infants from 1,501 to 2,500 Gm. of weight.

children. Frankenstein, who reported on the influence of diseases on the development of the immature infant, found that when diseases affected the growth of the immature infant they did so only during the first five years of life, and that after the fifth year the children overcame the delay due to illness.

Tables 8, 9, 10 and 11 also show that the lagging in growth is somewhat greater among girls than among boys. In agreement with Ylppö and Forschner-Böke, we also found that in general the smaller the weight at birth the greater the retardation in growth. Tables 9 and 10 show that those infants who weighed less than 1,500 Gm. at birth, generally remained the smallest in size. Among our cases, the children who weighed less than 1,500 Gm. at birth were on an average 2.7 cm. below the normal height, whereas those who weighed between 1,500 and 2,500 Gm. were on an average 0.58 cm. below the normal height; in other words, there is a definite relationship between lagging in growth and weight at birth.

The plotted heights of the individual immature infants are more below the normal line in the first seven years than in the last seven, as shown in the charts. Among the boys as well as among the girls, all but three infants in the first three and one-half years of life remained below the average growth curve. After that, some reached the normal line and others even went above it. After the seventh year, there is a slight tendency to crowding about the normal line, and in the case of the girls even to surpass it. But with few exceptions, the majority of the immature infants remain somewhat below the normal height up to, and in some cases, even beyond, the age of puberty.

Sitting Height.—We have also measured the sitting height of all our patients. The sitting height is the vertical distance between the surface on the top of the head and the surface on which the person is sitting. When the sitting height is subtracted from the height of a patient, the length of the extremities is obtained. The sitting height, after the second year can also be calculated by the following formula: sitting-height equals $\frac{\text{height}}{2}$ plus 5. I, therefore, calculated the sitting heights of normal children by means of the Camerer-Pirquet table and the foregoing formula. Figure 5 shows the curve of the sitting heights of normal children and also the plotted sitting heights of our immature children. One is immediately struck by the fact that the majority of the immature children, especially in the first ten years of life, have a sitting height that is higher than normal. Hence in comparing the standing height with the sitting height curve, we deduce that the delay in growth during the first ten years must be due, not to backwardness in the growth of the trunk, but to slow growth of the extremities. Normally, the extremities are relatively shorter than the trunk in children than in

TABLE 8.—*Retardation in Height and Weight in Immature Children*

Boys

	1-8 Years																									
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9		9-10		10-11		11-12		12-13		13-14		14-15	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	.....	.....	−1.5	+0.5	+1.5	−0.55	+4	−2 6	+10	−5.3	+7.5	−3.8	+12.5	+4.5	+8.5	−5.45	−1.5	−2.55	−2	−6.35	−5	−11	+24	−4	.....	.....	+15.5	−6.1
	....	.....	−5.5	−1.4	−1	−2.5	−2	−1.3	+12	+3.2	+9	−3.5	.....	.....	+10.5	−5.05	−0.75	−6.15	.....	.....	−1	−4.4	−9.5	−2.95	.....	.....	−5	−0.4
	.....	.....	−5	−1.72	−2	−0.3	−5	−1.5	−12	−0.6	−5	−2.53	.....	.....	−2.5	−4.6	.....	.....	.....	.....	.....	.....	+3.5	−3.10	.....	.....	.....	.....
	.....	.....	−5	−0.17	.....	.....	±0	−0.1	−4	−1.7	+9.5	−4.3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	−8.5	−5.75	.....	.....	.....	.....
	.....	.....	−8	−0.84	.....	.....	+8.5	+1.7	.....	.....	−3.5	−2	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	−10	−3.07	.....	.....	−2	−1.3	.....	.....	+3	−3.3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	−3.5	−1.13	.....	.....	−6.5	−1.3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	.....	.....	−5.5	−1.12	−0.5	−2.01	−0.43	−1.30	−2	−2.53	+3.4	−3.23	.....	.....	+5.5	−5.37	−1.12	−4.35	−2	−6.35	−3	−7.7	−4.8	−3.93	.....	.....	+5.25	−3.25

Girls

	1-8 Years														8-15 Years											
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9		9-10		10-11		11-12		12-13		13-14		14-15	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	−8	−0.5	−7	−1.5	−3.5	−1.55	−0.5	+0.3	+3.5	−2.6	−6	+1.15	+2	−2.1	+6.5	−3.4	+8	−1.5	−6.5	−1.2	−7	+5.3	+3	−3.85	+7	+8.8	−3	−2.1
	−5	−1.68	−2	−1.2	+7	−2.3	+3	+9.45	+3.5	+0.25	−1	−1.7	−2.5	−1.5	+1	−1.8	−3	+2.9	.....	.....	−7.5	−3.3	+7	−2.1	−5	−0.4	−5.5	−2.7
	−6	−0.95	−5	−1.95	+2.5	−2.45	+4.5	+0.8	−6	−0.75	−8	−0.7	+1.5	−.24	.....	.....	+1	−2.8	.....	.....	−8	−1.05	−5	+0.4	.....	.....	−5.5	+4.25
	−10	−0.3	−8.5	−1.5	+7.5	−0.95	−0.5	−1.75	−7.5	−2.05	.....	.....	.....	.....	.....	.....	+7	+5.2	.....	.....	.....	.....	+2	±0	.....	.....	−2.5	+0.05
	.....	.....	.....	.....	−5	−2.75	+2	−3.45	−3	+0.3	.....	.....	.....	.....	.....	.....	−17	+11.7	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	−6.5	−2.35	−2.5	−2.5	−5	−0.15	.....	.....	.....	.....	.....	.....	+1	+3.4	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	−3	−0.40	+3.5	+0.1	.....	.....	.....	.....	.....	.....	.....	.....	+10	+0.8	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	+7	−2.3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	−7	−0.68	−5.6	−1.54	−12 +0.8 −0.66	−1.58	+1.3	+0.42	−2.4	−0.83	−5	−0.42	+0.33	−2	+3.7	−2.6	+14	+2.81	−6.5	−1.2	−7.5	+0.31	+1.7	−1.38	+1	+4.2	−4.1	−0.15

* Hypophyseal disturbance.

General average of subnormaley in height of boys, −0.13 cm.; in weight, −1.08 Kg.

Average of subnormaley in height of boys from 1 to 8 years of age, −0.2 cm.; in weight, −3.29 Kg. (premature child).

Average of subnormaley in height of boys from 8 to 15 years of age, +0.6 cm.; in weight, −2.448 Kg. (premature child).

General average of subnormaley in height for girls from 1 to 15 years, −0.5 cm.; in weight, −0.979 Kg.

Average subnormaley for girls from 1 to 8 years: height, −0.5 cm.; weight, 1.89 Kg. (premature child).

Average of subnormaley for girls from 8 to 15 years: height, −0.45 cm.; weight, 2.0 Kg. (premature child).

TABLE 9.—*Retardation in Height and Weight of Immature Children with a Birth Weight under 1,500 Gm.*

Boys

	1-8 Years							8-15 Years						
	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15
	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.
	..... /	..... /	−2 / −0.3	..... /	..... /	−3.5 / −2	..... /	..... /	−1.5 / −2.55	..... /	..... /	+3.5 / −3.1	..... /	−5 / −0.4
Average	..... /	..... /	−2 / −0.3	..... /	..... /	−3.5 / −2	..... /	..... /	−1.5 / −2.55	..... /	..... /	+3.5 / −3.1	..... /	−5 / −0.4

Girls

	1-8 Years							8-15 Years						
	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15
	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.	Cm. / Kg.
	−5 / −1.68	..... /	..... /	..... /	..... /	..... /	..... /	..... /	+17 / +11.7	..... /	..... /	..... /	..... /	..... / .....
	−10 / −0.3	..... /	−5 / −2.75	+2 / −3.45	..... /	−8 / −0.7	−2.5 / −1.5	+6.5 / −3.4	+8 / −1.5	..... /	−8 / −1.05	..... /	−5 / −0.4	−5.5 / −2.7
Average	−7.5 / −0.99	..... /	−5 / −2.75	+2 / −3.45	..... /	−8 / −0.7	−2.5 / −1.5	+6.5 / −3.4	−4.5 / +5.1	..... /	−8 / −1.05	..... /	−5 / −0.4	−5.5 / −2.7

General average of subnormalcy in height for boys, —1.7 cm.; weight, —1.67 Kg.
Average of subnormalcy for boys from 1 to 8 years: height, —2.7 cm.; weight, —1.15 Kg. (premature child).
Average of subnormalcy for boys from 8 to 15 years old: height, —1 cm.; weight, —2.017 Kg.
General average of subnormalcy in height for girls, —3.1 cm.; weight, —0.987 Kg.
Average of subnormalcy for girls from 1 to 8 years of age: height, —3.5 cm.; weight, —1.577 Kg. (premature child).
Average of subnormalcy for girls from 8 to 15 years of age: height, —2.75 cm.; weight —0.408 Kg.
General average of subnormalcy for both boys and girls from 1 to 15 years of age: height, —2.7 cm.; weight, —1.188 Kg. (premature child).

TABLE 10.—*Retardation of Height and Weight of Immature Children with a Birth Weight from Between 1,501 to 2,000 Gm.*

Boys

	1-8 Years								8-15 Years							
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	.....		−1.5		−1		±0		−4		+9		.....		−2.5	
	.....		.....	−0.5	.....	−2.5	.....	+0.1	.....	−1.7	.....	−3.5	.....		.....	−4.6
	.....		−5.5		.....		+8.5		.....		+9.5		.....		.....	
	.....		.....	−1.4	.....	.....	.....	+1.7	.....	.....	.....	−4.3	.....	.....	.....	.....
	.....		−5		.....		.....		.....		.....		.....		.....	
	.....		.....	−0.17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....		−3.5		.....		.....		.....		.....		.....		.....	
	.....		.....	−1.13	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	.....		−3.9		−1		+4.2		−4		+9.2		.....		−2.5	
	.....		.....	−0.55	.....	−2.5	.....	+0.90	.....	−1.7	.....	−3.9	.....	.....	.....	−4.6

	8-15 Years													
	9-10		10-11		11-12		12-13		13-14		14-15			
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.		
	.....		−2		−5		+24		.....		+15.5			
	.....		.....	−6.35	.....	−11	*	−4	.....		.....	−6.1		
	.....		.....		−1		−9.5		.....		.....			
	.....		.....	.....	.....	−4.4		−2.95	.....		.....	.....		
	.....		.....		.....		−8.5		.....		.....			
	.....		.....	.....	.....	.....	.....	−5.75	.....		.....	.....		
	.....		.....		.....		.....		.....		.....			
	.....		.....	.....	.....	.....	.....	.....	.....		.....	.....		
	.....		−2		−3		−9		.....		+15.5			
	.....		.....	−6.35	.....	−7.7	.....	−4.35	.....		.....	−6.1		

Girls

	1-8 Years								8-15 Years							
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	−6		−5		.....		−2.5		.....		−6		.....		+1	
		−0.95		−1.95		.....		−2.5		.....		+1.15		.....		−1.8
	−8		.....		+2.5		+3.5		.....		.....		+2		.....	
		−0.5		.....		−2.45		+0.1		.....		.....		−2.1		.....
	.....		.....		−12		.....		−7.5		.....		.....		.....	
	.....	.....	.....	.....		+0.8	.....	.....		−2.05	.....	.....	.....	.....	.....	.....
	.....		.....		.....		.....		+3.5		.....		.....		.....	
	.....	.....	.....	.....	.....	.....	.....	.....		+0.25	.....	.....	.....	.....	.....	.....
	.....		.....		.....		.....		−3		.....		.....		.....	
	.....	.....	.....	.....	.....	.....	.....	.....		+0.3	.....	.....	.....	.....	.....	.....
Average	−7		−5		−4.7		+0.5		−2.3		−6		+2		+1	
		−0.72		−1.95		−0.82		−1.2		−0.5		+1.15		−2.1		−1.8

	8-15 Years													
	9-10		10-11		11-12		12-13		13-14		14-15			
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.		
	−3		−6.5		.....		.....		+7		−3			
		+2.9		−1.2		.....		.....		+8.8		−2.1		
	+7		.....		−7		.....		.....		−5.5			
		+5.2		.....		+5.3		.....		.....		+4.25		
	+1		.....		.....		.....		.....		−2.5			
		−2.8		.....		.....		.....		.....		+0.05		
	+10		.....		.....		.....		.....		.....			
		+0.8		.....		.....		.....		.....		.....		
	.....		.....		.....		.....		.....		.....			
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....		
	+3.75		−6.5		−7		.....		+7		−3.7			
		+1.52		−1.2		+5.3		.....		+8.8		+0.73		

* **Hypophyseal disturbance.**

General average of subnormalcy in height for boys, +0.2 cm.; weight, −2.167 Kg.
Average of subnormalcy for children from 1 to 8 years of age: height, +0.45 cm.; weight, −0.775 Kg.
Average of subnormalcy for boys from 8 to 15 years: height, −0.14 cm.; weight, −4.157 Kg.
General average of subnormalcy in height for girls, −0.74 cm.; weight, +0.313 Kg.
Average of subnormalcy for girls from 1 to 8 years of age: height, −1.88 cm.; weight, −0.512 Kg.
Average of hypernormalcy for girls from 8 to 15 years of age: height, +0.49 cm.; weight, +1.211 Kg. (premature child).
General average of subnormalcy for both boys and girls from 1 to 15 years ofage: height, −0.61 cm.; weight, −0.741 Kg. (premature child).

TABLE 11.—*Retardation in Height and Weight of Immature Children with a Birth Weight from Between 2,000 to 2,500 Gm.*

Boys

	1-8 Years								8-15 Years							
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	.....	.....	−8	−8.4	+1.5	−0.55	−2	−1.3	+10	−5.3	−5	−2.53	+12.5	+4.5	+8.5	−5.45
	.....	.....	−10	−3.07	.....	.....	−5	−1.5	−12	−0.6	+3	−3.3	.....	.....	+10.5	−6.05
	.....	.....	−5	−1.72	.....	.....	−6.5	−1.3	+12	+3.2	+7.5	−3.8	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	−2	−1.3	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	+4	−2.6	.....	.....	.....	.....	.....	.....	.....	.....
Average	.....	.....	−7.7	−1.88	+1.5	−0.55	−2.3	−1.6	−1	−2.95	+2.75	−3.21	.....	.....	+9.5	−5.75

	8-15 Years							
	9-10		10-11		11-12		12-13		13-14		14-15	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	−0.75	−6.15	.....	.....	.....	.....	−8.5	−5.75	.....	.....	−5	−0.4
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	−0.75	−6.15	.....	.....	.....	.....	−8.5	−5.75	.....	.....	−5	−0.4

Girls

	1-8 Years								8-15 Years							
	1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-9	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	.....	.....	−7	−1.5	−3.5	−1.55	+3	+9.45	+3.5	−2.6	−1	−1.7	+1.5	−2.4	.....	.....
	.....	.....	−2	−1.2	+7.5	−0.95	+4.5	+0.8	−6	−0.75	.....	.....	.....	.....	.....	.....
	.....	.....	−8.5	−1.5	−3	−0.4	−0.5	−1.75	−5	−0.15	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	+7	−2.3	−0.5	+0.3	.....	.....	.....	.....	.....	.....	.....	.....
	.....	.....	.....	.....	−6.5	−2.35	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	.....	.....	−5.8	−1.4	+0.3	−1.51	+1.6	+2.2	−2.5	−1.17	−1	−1.7	+1.5	−2.4	.....	.....

	8-15 Years											
	9-10		10-11		11-12		12-13		13-14		14-15	
	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.	Cm.	Kg.
	.....	.....	.....	.....	−7.5	−3.3	+7	−2.1	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	+2	±0	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	+3	+3.85	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	−5	+0.4	.....	.....	.....	.....
	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average	.....	.....	.....	.....	−7.5	−3.3	+1.75	−1.36	.....	.....	.....	.....

* Hypophyseal disturbance.

General average of subnormalcy in height for boys, −0.60 cm.; weight, −1.485 Kg.
Average of subnormalcy for boys from 1 to 8 years of age: height, −0.48 cm.; weight, −0.728 Kg. (premature child).
Average of subnormalcy for boys from 8 to 15 years of age: height, −0.94 cm.; weight, −3.61 Kg. (premature child).
General average of subnormalcy in height for girls from 1 to 15 years of age, −0.53 cm.; weight, −0.7 Kg.
Average of subnormalcy for girls from 1 to 8 years of age: height, −0.35 cm.; weight, −0.352 Kg. (premature child).
Average of subnormalcy for girls from 8 to 15 years of age; height, −1.15 cm.; weight, −0.932 Kg. (premature child).
General average of subnormalcy for both boys and girls from 1 to 15 years of age: height, −0.56 cm.; weight, −1.062 Kg.

adults. It is, therefore, conceivable that whatever factor makes for comparative delay in growth of the extremities in children is still more potent in immature children. I, therefore, disagree with Rosenstern who says that the relatively short extremities of the immature infant disappear after the third year of life. On the contrary, in the series studied I found that the delay in the growth of the extremities of the immature child persists until about the tenth year, and that this is the principal cause of the retardation in the height of the immature child.

Weight.—What has been said regarding lagging behind in the height in immature children is still more applicable to their weight. Ylppö said that his infants, especially those with a birth weight under

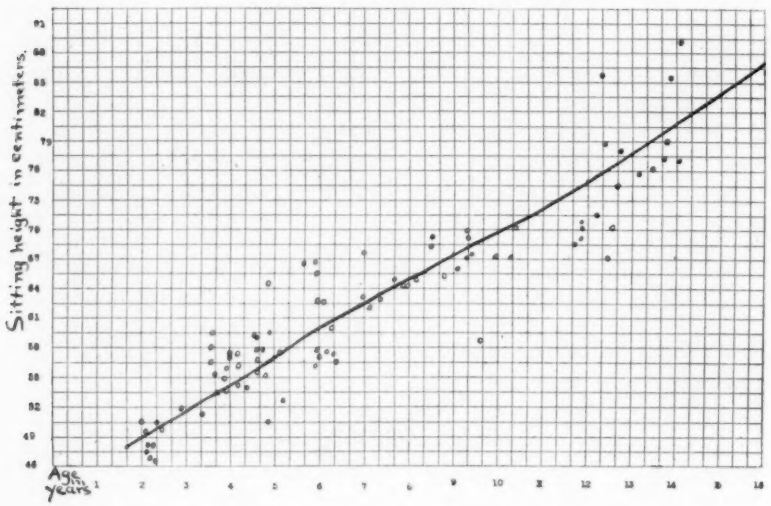

Fig. 5.—Curve of normal sitting height with sitting heights of immature infants.

1,500 Gm., attained normal weights between the fifth and sixth years of life. Forschner-Böke found that in her cases the infants did not attain the normal weights even at the age of 7. We found that the great majority of cases are below the normal weight standard even at the ages of from 14 to 15. Figures 6 and 7, which show the comparison of the weights in the series studied with the normal Camerer-Pirquet standard, bring out this fact strikingly. The figure shows that the boys are more subnormal than the girls. Of all the male immature children (four cases were not plotted because they represented hypophyseal disturbances), only one is above the normal line. This is quite conceivable, as we know that boys lag behind in growth less than girls, and therefore their weight is too little for their height. While the average boy in our cases was over 1 Kg. underweight, the average girl was only about

80 Gm. subnormal. The smaller the weight at birth the longer the subnormality in weight persists. In brief, immature children, especially boys, are more subnormal in weight than in length, and children with a birth weight under 2,000 Gm. show a greater difference between the actual and standard weight than those with a weight at birth of between 2,000 and 2,500 Gm.

Skull.—In the immature infant, there is a constant growing and enlarging of the large and small fontanels and a widening of the sutures during the first three to four months of life. This is due to the development of the megacephalus and the early rachitic disturbances. In the majority of cases, craniotabes is also already pronounced at this time, and the skull bones have then a leather-like consistency. This almost

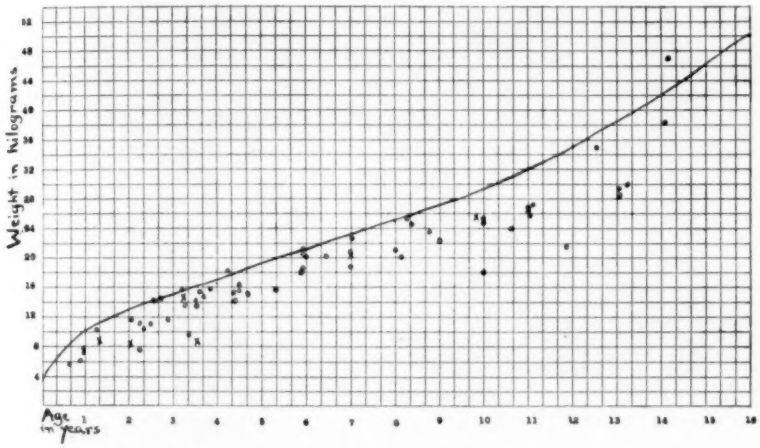

Fig. 6.—Weight of immature male infants in comparison with normal weight curve based in Camerer-Pirquet figures. The crosses indicate infants under 1,500 Gm. of weight at birth; the zeros, infants from 1,501 to 2,500 Gm. of weight at birth.

constant occurrence of osteomalacia of the skull in the immature infant begins to disappear at the beginning of the third trimester of the first year of life and continues to improve slowly, so that complete hardening occurs about the middle of the second year. This osteomalacia leads to the so frequent occurrence of deformities of the skull in the immature infant, which in some cases even persist into later life. A strikingly large number of our infants showed such deformities of the skull. Some still showed a flattening of the occiput, others a protruding forehead, while the majority of them had parietal, occipital or frontal tuberosities of small or moderate degree. There were also five cases of microcephalus, three of megacephalus and one of hydrocephalus.

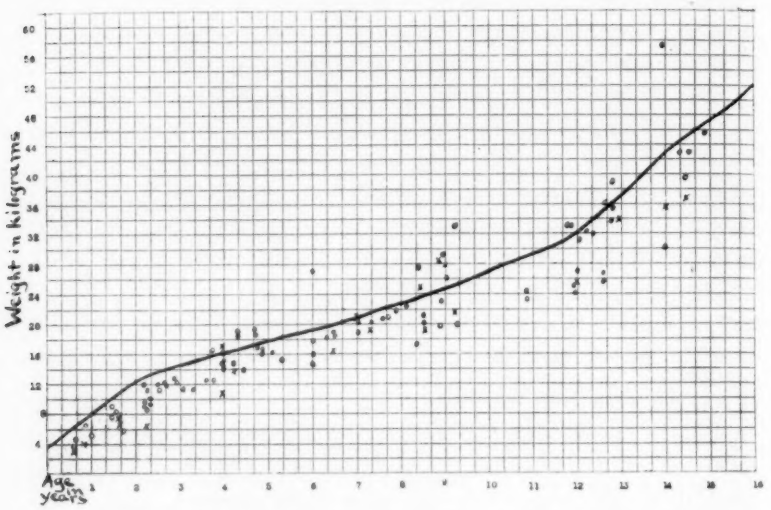

Fig. 7.—Weight of immature female infants in comparison with normal weight curves, based on Camerer-Pirquet figures. The crosses indicate infants under 1,500 Gm. of weight; the zeros, infants from 1,501 to 2,500 Gm. of weight.

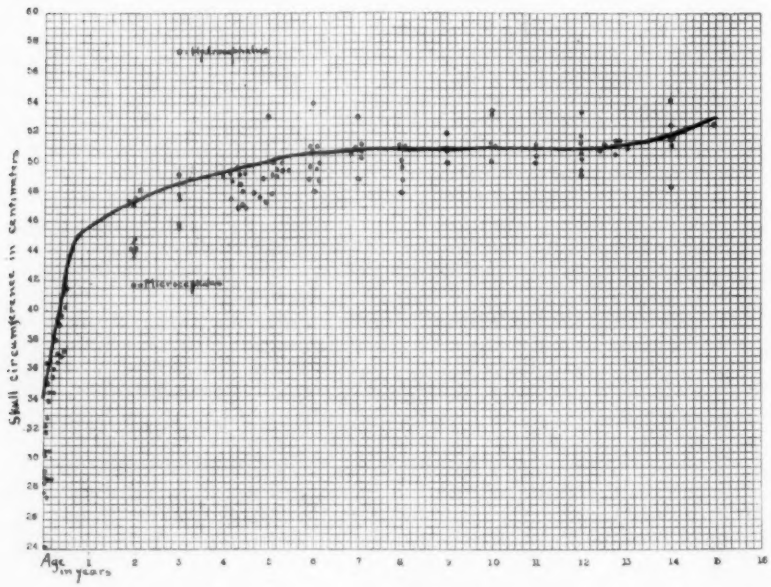

Fig. 8.—Growth of the skull of immature infants in comparison with the growth of the skull of mature infants according to the figures of Gundobin and Monti.

Table 12 gives our average figures of the measurements of the skulls of immature infants from 2 to 15 years of age. The measurements were made through the greatest fronto-occipital circumference. Gundobin's figures for mature infants are given beside our figures for comparison. This table and figure 8 show that the circumference of

TABLE 12.—*Comparison of Figures on Circumference of the Skull*

Age	Size of Skull	
	Gundobin's Figures for Mature Children	Our Figures for Immature Children
New-born infant	34.2	....
3 months	38.9	....
6 months	41.9	....
9 months	44.5	....
1 year	46.4	....
2 years	48.1	47.4
3 years	49	48.6
4 years	49	48.8
5 years	50.1	48.9
6 years	50.8	49.5
7 years	50.8	49.9
8 years	51	49.9
9 years	51	49.9
10 years	51	50
11 years	51	50.7
12 years	51	51.2
13 years	51	51.3
14 years	52	52.3
15 years	53	53

TABLE 13.—*Comparison of Figures on Circumference of the Chest*

Age	Size of Chest	
	Gundobin's Figures for Mature Children	Our Figures for Immature Children
New-born infant	32	....
3 months	37.5	....
6 months	40.3	....
9 months	43	....
1 year	45.8	....
2 years	49	45.4
3 years	50.6	49.6
4 years	52.6	51.3
5 years	54	55
6 years	56	56.5
7 years	58	58.7
8 years	59	60
9 years	61	61.7
10 years	62	63.3
11 years	62	65.6
12 years	65	65.6
13 years	66.5	68.9
14 years	67.5	75
15 years	70.5	77.5

the skull of the immature child, up to 12 years of age, remains slightly below that of the mature child, and that afterward it even overtakes it somewhat. Figure 8 also shows that up to 6 years of age the circumference of the skull remains more behind that of the normal than during the later years of life.

Frankenstein, who studied the influence of diseases on the growth of the immature child, said that infectious diseases do not influence the

growth and development of the skull. The two factors that are most responsible for the deviation of the immature skull from the normal are the megacephaly and rickets. The megacephaly, dependent on the growth of the brain independently of the body, makes for a relatively large skull when the child is between 3 and 18 months of age. Rickets, by its early development in the immature infant (first three months of life) leads to osteomalacia, which in turn easily affects the shape of the skull, temporarily at least. In some of the cases, these rachitic deformities and in a few even the megacephalus persist to and beyond puberty.

Chest.—The figures for the circumference of the chest were obtained by measuring the chest through the nipple line at the end of expiration.

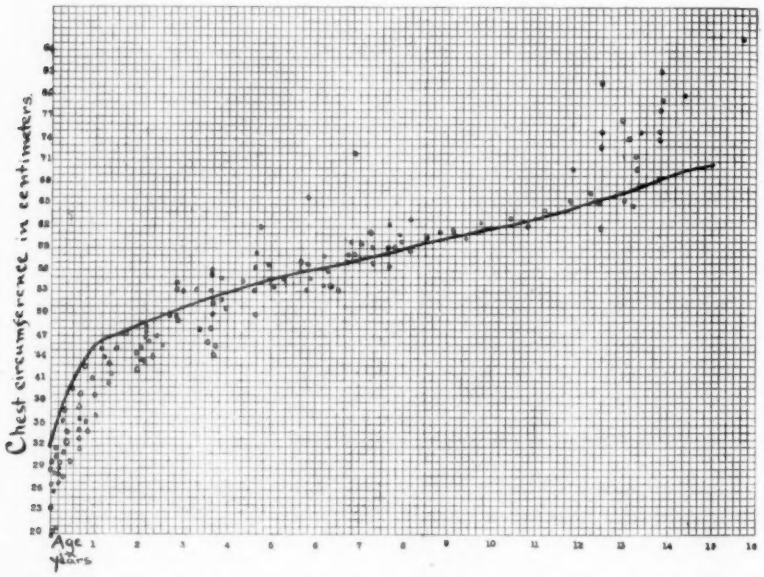

Fig. 9.—Growth of chest of immature infants in comparison with that of a mature infant according to Gundobin's figures.

Table 13, in which the figures from the clinic records are beside those of Gundobin for mature children, and figure 9 show that the circumference of the chest of the immature child up to the fifth year remains below that of the mature child; this is especially true during the first two years of life. In the first two years, there is active growth of the chest, and in a few cases it has increased by one half of the original circumference. After the sixth year, the chest of the immature child assumes larger proportions than normally and continues to remain above the normal line until past puberty. What the causes for this overgrowth of the chest could be, I am unable to say at present, unless they be the rachitic changes—thickened costal epiphyses, falling in of the flanks,

and rachitic enlargement of the sternum—which Ylppö regards as the cause of the large diameter of the chest in his cases at from 1½ to 1¾ years of age. In our cases, the crossing occurs much later, namely, between the second and the third years. Comberg also obtained higher figures than Ylppö in her measurements of chests of immature children.

Pelidisi.—The foregoing considerations show that the height and weight of the average immature child are below the average normal height and weight; the latter is more below normal than the former. It becomes of interest to determine whether the child that is subnormal in height and weight is proportionately constituted; i. e., it is of interest to have an objective measure of the state of nutrition of the immature child. Pirquet measures this state of nutrition by a determination of

TABLE 14.—*Pelidisi Determinations*

Pelidisi	Number of Boys	Number of Girls
110	..	1
104	..	1
102	2	0
101	0	2
100	0	4
99	1	5
98	4	5
97	3	4
96	2	7
95	8	11
94	7	13
93	2	10
92	10	7
91	4	6
90	8	6
89	6	6
88	1	4
87	3	2
86	0	4
85	1	1
Total	62	99

the relation existing between the sitting height and the body weight. He found that the sitting height raised to the third power is equal to ten times the body weight, or expressed mathematically Si^3 equals $10 \times$ weight or the cube root of ten times the weight of the infant is equal to the sitting height, thus $\sqrt[3]{10 \times \text{weight}} = Si$, dividing both sides by Si, $\sqrt[3]{\frac{10 \times \text{weight}}{Si}} = 1$ or 100 degrees pelidisi (pondus decies, lineare, divisio, sedentis altitudo). The pelidisi index is 100 in healthy muscular adults and in fat infants. The results of extensive measurements on school children yielded the facts that children with a pelidisi index under 94.5 were undernourished, from 94.5 to 100 normally nourished and over 100 overnourished.

We have determined the pelidisi index of our patients, and the results are seen in table 14. Of the sixty-two boys, two are overnourished, eighteen are normal and forty-two, or 68 per cent, are undernourished, i. e., the pelidisi index is under 94.5. Of the ninety-nine girls,

eight are overnourished, thirty-two are normal and fifty-nine, or about 59 per cent, are undernourished. Hence over two thirds of the boys and over one half of the girls are undernourished. This corresponds also to our clinical impression. There were few well proportioned and well nourished types among the patients, especially among the boys. A large number of them belonged to the asthenic type. They were thin children, had a long trunk, a little or moderate deposit of subcutaneous fat, thin arms and thin legs, and not much musculature. Among the girls there were a few short, fat, compact and chubby ones, which also corresponds to the objective pelidisi observations.

Conclusions.—I thus arrived at the following conclusions with regard to the physical development of the immature infant:

1. The immature infant remains behind the normal both in increase in height and in weight up to and even beyond puberty.

2. He is more subnormal in weight than in height.

3. The smaller the weight at birth, the greater the retardation in the gain in weight and in height.

4. Girls lag more behind in gain in height, while boys lag more in gain in weight.

5. The diameters of the chest and skull are also smaller than normal, the former until the sixth and the latter until the twelfth year; but this delay in growth is only transitory.

6. Since the majority of the immature infants belong to a definite type, namely, the asthenic type, it is likely that their backwardness in gaining in height and in weight, since it has not been overcome at puberty, is not transitory but will remain as a permanent condition of physical subnormality.

MENTAL DEVELOPMENT OF THE IMMATURE CHILD

General Considerations.—Striking as the retardation in the physical development of the immature infant may be, one is still more impressed with the multitude of deviations from the normal that one finds in reviewing the psychic or mental progress of these children. This mental subnormality is evidenced even in the first year of life by the late development of static functions, such as holding up the head and attempts to sit up. In the second year, it comes to expression in the late attempts at walking or active speaking. Unfortunately, these are not all. As the child is followed in its mental unfolding, it is found that some do not keep up the normal progress in school with the rest of their mature fellow children; others must be sent to schools for mentally inferior children, while a few never attain a sufficient degree of intelligence to attend school. Of the large number that seems normal, a

great many are or become neuropaths. In brief, the immature infant will become the backward school child, is the potential future psychopathic or neuropathic patient and even the potential inmate of the home for imbeciles or idiots.

In order to obtain dependable data regarding the mentality of immature children, I tried whenever possible to compare such a child with its normally mature brother or sister. Inquiry was always made as to the age when the child first developed the static functions, i. e., sitting, standing and walking; when the child ceased bed-wetting; and, finally, when the first attempts at speaking were made. We realize, of course, that the information thus supplied by the mother was not always reliable, but when a larger number of cases is under consideration, the error will not be so great. The smaller children were then tested in the recognition of striking colors, such as white, black and red, in counting, in naming the street that they lived on, in naming their brothers and sisters and in naming common objects. The older children were questioned about their grades in school and about arithmetic, reading, recitation and names, and the ones who were very much backward in school or who seemed mentally inferior were subjected to regular intelligence tests.

Static Functions.—Figure 10 shows that some immature children began to sit up during the fifth month, but that others first sat up between from 9 to 18 months of age and two at 30 months of age. Children with a birth weight from between 2,000 to 2,500 Gm. began to stand at eight months, but of those with a birth weight under 2,000 Gm., none stood up before the twelfth month. Some of the children did not learn to stand until the third year of life. Attempts at walking were first made by a few of the children at nine months; by the majority of the children they were made between the twelfth and twenty-fourth month, but some (seven infants) did not begin to walk until the thirty-sixth month; three did not begin to walk until the end of the fourth year. Brandt's figures concerning the time when his immature children learned to walk are: end of first year, fifteen children; end of fifteen months, twenty children; end of eighteen months, fifteen children; end of twenty-one months, thirteen children; end of two years, twenty-three children; end of two and one-half years, three children; end of three years, one child, and over three years, five children. The delay of the beginning of the static functions is variable and also considerable, but in some of the cases it may be due not so much to a tardy nervous development as to rachitic changes which occur so frequently and so early in immature children.

Speech.—Nadoleczny considered from 8 to 9 months of age as the normal time for repeating words, 15 months for active speaking,

and 2 years for sentence making. The immature children of our series began to speak at 12 months, a large number of them began at 18 months, ten began at 36 months, three at 4 years, and one at 4½ years of age.

A number of the children showed defects in speech, but they were all in children under 8 years of age; Ylppö said that in most cases these defects in speech disappear in time. Seven children were lispers, two

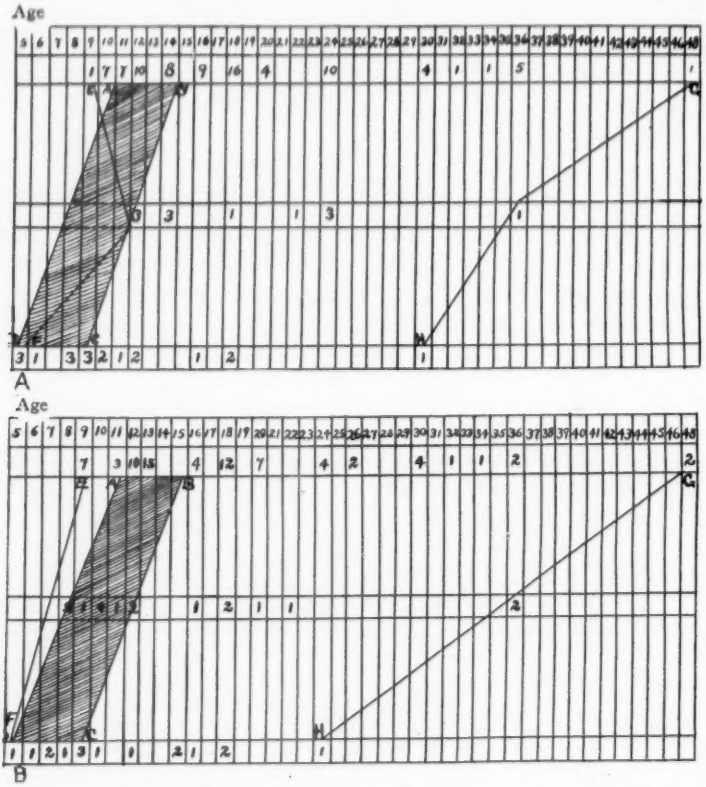

Fig. 10.—*A* shows the age in months at which static functions began in immature infants under 2,000 Gm. of weight. The shaded area *ABCD* represents the normal physiologic variation during which the static functions begin; area *EFGH*, the period during which the static functions begin in the immature child. *B* shows the age in months at which the static functions began in immature infants between 2,001 to 2,500 Gm. of weight. The shaded area *ABCD* represents the normal physiologic variation during which the static functions begin; area *EFGH*, the period during which static functions begin in the immature child.

stutterers, one had a marked nasal speech and four had difficulty in pronouncing certain letters, especially the L and the N. Altogether, there were fourteen of ninety-seven children, or 15 per cent, who had a defect in speech. In addition, there were two microcephalic idiots in our series who could say only two or three syllables and one deaf-mute.

School.—Of fifty-one immature children of school age that presented themselves for reexamination, four had Little's disease associated with idiocy or imbecility; one was an idiot, and one child was of age but too small to attend school. Of the remaining forty-five children, fifteen, or only one third, were in classes that properly corresponded to their ages. Twelve children, or about 27 per cent, were one year behind in their class, nine, or 20 per cent, were two years behind, two were three years behind and two were four years behind in their class in school. Five children, or over 11 per cent, were attending schools for mentally inferior children. Of the fifteen that were not behind in their school work, only one was farther ahead in his school work than his schoolmates; all the others were average pupils and students.

Brandt reports that nine of fifty-three immature children, or 17 per cent, were one year behind in school. He also says that thirty-six children, or 50 per cent, of the immature children that came for reexamina-

TABLE 15.—*Age in Months at Which Immature Children Began to Talk*

Age	12	14	16	18	20	24	28	30	31	48	54
Number of children	35	15	9	28	7	22	3	6	10	3	1

tion gave the impression of being neurotic. Of his seventy-two cases, eight made a wideawake mental impression, forty-seven made a normal average mental impression, four were mentally sluggish, four were psychopathic, six were mentally inferior and three had Little's disease. In brief, the immature child is a definitely backward pupil and swells the ranks of mentally inferior children.

Mental and Nervous Disturbances.—A closer review of our cases brings to light an array of psychopathic and neuropathic children that will rarely be found in another group of 100 children. There were in our series five children, or 5 per cent, who suffered from epilepsy, one with epilepsy of the jacksonian type, one with petit mal and three with grand mal. Schwartz said that his anatomic studies showed that epilepsy can in many cases be due to typical cerebral injury at birth; cortical injuries, and particularly the lesions of Ammon's horns deserve especial mention. Edinger had already shown that injury of Ammon's horns at birth must be considered as a possible cause of epilepsy. We found five cases of enuresis; the patients were: a girl aged 4, a boy aged 8, a girl aged 8 years and 2 months, a boy aged 19; there was also one case of diurnal enuresis in a boy aged 6 years and 11 months. Two children of our series, a boy and a girl, were brought to the hospital by the mother because they masturbated. One girl aged 6½ years was a deaf-mute. One boy aged 11 years and 1 month was a night walker.

There were sixteen, or about 16 per cent, definitely neuropathic children. In some the neuropathy was of the digestive type; their symptoms were anorexia, nervous vomiting, spastic constipation and fluctuation in body weight. Several were of the asthenic type; they were pale, had cool perspiring extremities, were subject to headaches and showed evidence of vasomotor instability. There was one case of motor neuropathy with hypertonicity and hypermotoria, the latter so marked that the movements almost seemed choreiform. All of these children had increased patellar reflexes. Besides these definitely neuropathic children, a large number of the older children showed individual symptoms of neuropathy, such as pavor nocturnus, being easily frightened, forgetful and absentminded; they cried on the least provocation and had fits of madness. In many cases the mothers complained that the children seemed mentally weak; they had to be helped with their school work and were unable to concentrate in the school, so that the parents had been asked to come to see the teacher on several occasions. A large number of the children seemed to have especial difficulty in mathematics. In one of the immature cases, a boy aged 19 years and 7 months had finished only the fifth grade at the age of 14; he had been apprenticed to a plumber, but had to give it up because it was too difficult for him to learn, and he is now working in a paper-box factory where he is doing the simplest possible work. His intelligence is that of a boy aged 12; in other words, he is a moron. Pototzky, who studied the immature outpatients of the Auguste Viktoria Hospital in Berlin, said that especially immature children develop psychopathic and intellectual disturbances as well as disturbances of will and emotion which makes it difficult to rear and to educate them.

Bondi said that while a large part of the premature infants do not show any signs of inferiority in later life, in full term but small infants he saw serious defects, such as neuropathy and delayed growth, which were especially striking when these children were compared with normal children. Schwartz, in his large material observed at autopsy, found cases of porencephaly due to hemorrhages that had occurred during birth. He concludes then that the pathologic condition of the new-born infant is based primarily on cerebral injuries at birth, and that epilepsy, idiocy and Little's disease are the end-results of cerebral trauma at birth. He found that the older the hemorrhage, the greater was the cerebromalacia and the greater the number of secondary scars. But even in children with cerebral trauma at birth who lived only from two to four days, he found macroscopically visible focal areas of softening in the brain in the majority of the cases.

Of 2,000 cases from the neurologic pediatric clinic of Boston, Friedman reported 266, or 13.3 per cent, cases of nervous and mental disturbances due to cerebral trauma at birth. Pfaundler found in his

material of immature children 8.3 per cent mentally well developed, and 2.2 per cent definitely imbecile. Schwartz, in America, did not discover any evidence of mental defect in his cases of premature and immature children. Also Comberg, who reexamined fifty immature children, did not find any evidence of mental inferiority among them. Ylppö found severe idiocy or imbecility in 7.4 per cent of his material, and Little's disease in 3.1 per cent of the cases. He said that the occurrence of Little's disease and idiocy would be still more frequent among immature children were it not for the fact that many of those who would have these diseases die from cerebral trauma a few days after birth. We found five cases, or 5 per cent, of Little's disease among our children, a strikingly large number. One of the patients was a boy, aged 11 years and 8 months, who had a spontaneous birth and whose weight at birth was 1,900 Gm. He was also an imbecile and had convulsions until the sixth

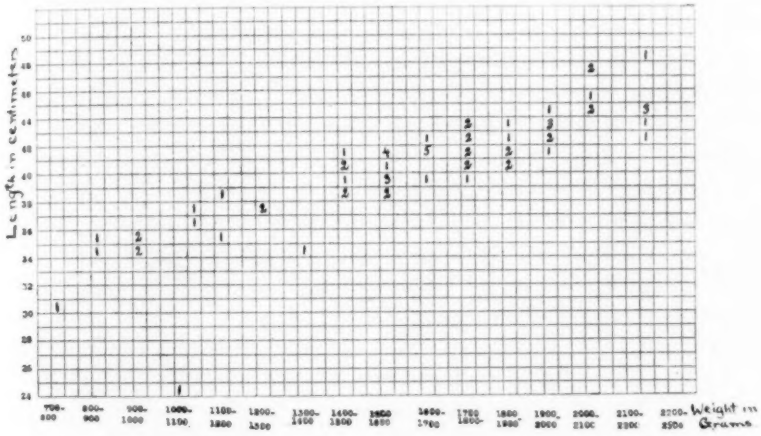

Fig. 11.—Review chart showing in graphic form the number of immature infants with their length and weight. Note that the majority are from 1,420 to 2,100 Gm. in weight, and from 38 to 45 cm. in height.

year of life. The second case was that of a boy, an idiot, who also suffered from left-sided jacksonian epilepsy. He was born by a head presentation, with forceps delivery, and weighed 2,000 Gm. The third case, that of an imbecile boy, aged 6 years and 8 months, weighed 2,300 Gm. at birth and was born by a breech presentation with a forceps delivery. The fourth boy, aged 8 years and 3 months, an imbecile, also suffered from epileptic convulsions. He was born by a forceps delivery and weighed 1,950 Gm. The fifth patient was also a boy, who weighed 2,085 Gm. at birth, and was delivered by cesarean section. All of the boys weighed under 2,300 Gm.; three were first born children, and three of them were delivered by forceps. Intelligence disturbances do not necessarily occur in Little's disease, although as a rule imbecility

or idiocy accompanies the disease. Besides the two idiots and two imbeciles who suffered with Little's disease, we had in our series two more idiots and one moron, altogether seven cases of severe mental disturbances, or 7 per cent.

Some infants die a week or so after birth as a result of sepsis or bronchopneumonia, and at postmortem examination one is surprised to find an intracranial hematoma. It is, therefore, possible that small hematomas may remain latent, and only after organization and scar formation do they give signs of epilepsy or Little's disease. In this manner may be explained certain cases of epilepsy or Little's disease in which the postnatal course was normal. Furthermore, it is not always necessary—especially in the case of immature infants—that difficult labor or delivery by forceps should precede the development of severe cerebral disturbances. One of our infants who had Little's disease was delivered by cesarean section. This is explained on the basis of the marked vasolability of the immature infant, so that only slight trauma—merely uterine contractions—may bring about intracranial injury; in other words, the intracranial hemorrhage in many of the immature infants is due not so much to the mechanical trauma associated with the delivery as to the delicateness and embryonic state of the capillary system. This is easy to understand, since the new science of capilloroscopy has taught that in the immature child there is a persistence of the delicate fetal capillaries or the archicapillaries. These are present in the brain as well as in the cutis, and it takes little force to injure these delicate and poorly elastic capillaries.

Denis, in 1826, Billard, in 1828, and Cruveilhier, in 1829, first mentioned the possible relationship between intracranial hemorrhage at birth and subsequent cerebral spastic paralysis. In 1843, Little stated in his first paper that cerebral spastic paralysis in children was due to underdevelopment of the nerve tissues and to intracranial meningitis. Nineteen years later, in 1862, he made the statement that about 75 per cent of the cases of cerebral spastic paralysis in children were caused by intracranial hemorrhage at birth. In 1885, McNutt confirmed this opinion by postmortem studies. Since then, a number of investigators have studied the subject and confirmed the causal relationship between cerebral trauma at birth and cerebral spastic paralysis. The most notable contributors to this subject were Beneke (1910), Pott (1911), Green (1914), Mayer (1915), Brady (1918), Warwick (1919), Holland (1920), Rodda (1920), Sidbury (1920), Schaefer (1921), Schwartz (1922), Capon (1922), Huenekens (1923), and Sharpe and MaClaire (1916, 1918, 1924 and 1925). The last authors say that:

> They examined both at operation and at autopsy the brains of a large series of chronic cases of cerebral spastic paralysis, and the pathology has been practically the same—"wet" oedematous brains under varying degrees of increased

pressure, and along the supracortical veins in the sulci has been demonstrated the cloudy, whitish, new tissue formation with a fibrinous thickening of the walls of these veins—the organization residue of a former layer of supracortical hemorrhage which occurred most probably at the time of birth, and of larger amount than could be entirely absorbed by the natural means of excretion. Since it has now been demonstrated that over 80 per cent of the cerebro-spinal fluid is normally excreted through the walls of the supracortical veins, the partial blockage of this main channel of excretion of cerebro-spinal fluid by the organization residue of a former hemorrhage in these patients, explains the "wet" oedematous condition of the brain under varying degrees of increased pressure, and the resulting spastic paralysis in its various forms associated with a mental retardation of a varying degree.

Sharpe's examination of 5,192 patients with various forms of cerebral diplegia, between the ages of 34 days and 23 years, yielded the following data: 81 per cent were first born children; 95 per cent were mature infants (but it is not stated whether or not they were of low birth weight); 90 per cent were born after a prolonged and difficult labor; 76 per cent were born after forceps delivery, and 17 per cent after breech delivery. During the first few weeks, 64 per cent of these children were more somnolent than normally; 23 per cent refused the breast; 78 per cent did not have a normal desire for food; 39 per cent had twitchings of various grades; 17 per cent had general convulsions in the first two weeks of life; 61 per cent were declared to be normal at the end of the first fourteen days and 82 per cent at the end of the first month. In 79 per cent of the cases, the first signs of the disease became manifest during the last months of the first year of life. In 671, or 12 per cent, there were definite changes of increased intracranial pressure leading to ophthalmoscopic changes.

Although Wall claimed that he did not see Little's disease, idiocy, imbecility and epilepsy any more frequently among premature children than among other children, he admitted that a large number of them show delayed mental development, and "that these children learn to talk and walk later, remain enuretics a long time, and make only mediocre progress in school; but this tardy development is not a permanent or irreparable one."

Schwartz said that the old encephalitis interstitialis of Virchow is a process of degeneration based on injuries received during birth. Fatty processes, degeneration and infiltration of the brain of new-born infants, focal as well as diffuse, are in the overwhelming majority of the cases caused by traumatic injuries during birth, and the foregoing changes represent secondary processes. Then follow cerebromalacia, scarring and sclerotic processes. The malformations of the brain that may be secondary to trauma at birth are: (1) peripheral and central porencephaly, (2) sclerosis, and occasionally internal hydrocephaus.

We may, therefore, conclude that cerebral injury at birth is one of the most common causes of congenital mental inferiority.

DEVELOPMENT OF PUBERTY

While immaturity has an injurious effect on the physical and mental development of the child even up to the age of puberty, we did not find signs of any deleterious influence on the development of puberty in immature children. We had sixteen children in our series between the ages of 11 years and 10 months and 19 years and 7 months, thirteen of whom were girls. All the girls had enlarged mammary glands, and all but two had axillary and pubic hair. Seven of the thirteen girls had not yet menstruated, the oldest one of these being 14 years of age; the average age of the nonmenstruating group was 13 years and 5 weeks. Of the six girls that were menstruating, the earliest age at which the periods occurred was 12 years and 5 months, and the latest, 14 years and 2 months; the average age of onset of menstruation was 13 years and 5½ months. The ages of the three boys in this group were 19 years and 6 months, 14 years and 2 months and 12 years and 3 months, respectively; all had axillary and pubic hair, well developed external genitalia corresponding to their ages and bearded faces. The boy aged 12 years and 3 months, who will be mentioned later, in the discussion of hypophyseal disturbances, had strikingly large genitalia corresponding to those of a mature man, had a definite beginning of a moustache and a heavy growth of pubic hair. All the patients, therefore, at the age of puberty showed the secondary sexual characteristics corresponding to their ages, and there were no signs evident of delay or retardation in the development of the characteristics of puberty that could be interpreted as being due to the immature birth.

THE INFLUENCE OF ACUTE AND CHRONIC DISEASES ON THE DEVELOPMENT OF THE IMMATURE CHILD

Infections in the immature child are of much more serious consequence than in mature children. This is especially true in the case of infections that occur during the first weeks of life. The hematopoietic system, like the other organs of the body, is incompletely developed, and this therefore lessens the capacity of the body to resist and to localize infections. It is due to this fact, together with the general poor resistance of the young infant, that infections which in an older child run a mild course assume in the new-born infant, and especially in the immature infant, threatening proportions and often terminate in general sepsis. Ylppö demonstrated that in immature infants suffering from enteritis it is not uncommon for bacteria to pass through the intestinal wall, enter the blood stream and cause sepsis; also, suppurative parotitis secondary to enteral infection, hardly ever seen in older children, is not uncommon in immature infants. On the basis of blood examinations, Delestre showed that in immature infants bacteria often circulate in the blood stream; in contradistinction to Ylppö, he assumed that the lungs

are the most common point of entry. Ylppö also showed that those forces which bring about the formation of fibrin and purulent exudates in peritonitis and pleuritis in full term infants are only slightly or not at all developed in immature infants, and the mentioned infections manifest themselves through serous exudates. According to Ylppö, grip in immature infants, instead of taking the course of a respiratory infection, leads to an enteritis which is usually followed by death. Serious as these acute infections may be for the immature infant, however, all do not terminate fatally. We have had several cases of infection in the immature infants, chiefly bronchopneumonia and furunculosis, which ended in recovery. Similar cases are reported by Comberg and Ylppö.

Experiments on the bactericidal capacity of the blood of immature infants showed that the bactericidity is present and is just as well developed in the first days of life in the immature infant as in the full term infant. These experiments further showed that various diseases, with the exception of acute nutritional disturbances, do not have an unfavorable effect on the bactericidal capacity of the blood, but that acute nutritional disturbances bring about a lowering of the bactericidal power in almost all cases, which indicates that bactericidity is more readily injured in immature infants than in full term infants.

Frankenstein found that the diseases which contribute most to delayed growth in the immature infant are, with the exception of rickets, first and foremost, grip with or without pneumonia, and second, acute and chronic nutritional disturbances. He concluded by saying that "while those immature infants whose first months of life were without exogenous influence developed, in the majority of cases, into healthy children, the majority of ill immature infants form a definite type of sickly or predisposed individuals."

Bronchopneumonia was found at autopsy in sixty-five of 117 of our patients who died during the first month of life. It was the only condition found in forty-one. Those who died during the first few days of life showed chiefly the hemorrhagic form of bronchopneumonia. This shows that respiratory infections are deadly diseases in new-born infants, especially in immature infants, and too much care cannot be exercised in the prophylaxis of respiratory infections in a ward for immature infants.

Rickets is almost a constant occurrence in immature infants; in fact, if signs of rickets are discovered in an infant from 6 to 8 weeks of age, the case can be diagnosed almost with certainty as one of prematurity or immaturity. Ylppö said that from four to eight weeks after birth one finds roentgenologic evidence of rickets in these infants. I have spoken previously of the early craniotabes which leads to deformities of the head; the chest also suffers marked deformation from early

rickets. Of our patients who came for reexamination, forty-eight showed more or less of a rosary, nineteen had genu valgum or varum and ten had either a funnel or pigeon chest or a marked flaring out of the lower costal border. About one half of the immature infants, therefore, on reexamination showed signs of healed rickets as follows: tubera frontalia et occipitalia, twelve cases; funnel or pigeon chest or flaring, ten cases; rosary, forty-eight cases; genu valgum or varum, nineteen cases, and lordosis or kyphosis, three cases.

In view of the almost constant appearance of rickets in immature infants, it is advisable to treat the children prophylactically as soon as possible after birth. As soon as the body temperature is no longer inconstant, one may begin to expose the child to the quartzlight in a well heated room. It is best not to give anything by mouth as it may upset the delicate stomach, unless ergosterine (which at present is the subject of experimental work by various investigators) proves to be efficient, and at the same time does not have an unfavorable influence on the digestion of the child.

TABLE 16.—*Dentition in Immature Children*

Age in months	6	7	8	9	10	11	12	13	14	15	16	18	20	24	36
Number of children	24	17	24	16	12	9	16	5	11	1	3	7	1	4	1

Dentition was delayed in a large number of the children owing to rickets. A number of children also showed considerable dental caries, especially of the incisors; we were, however, unable to determine whether or not the caries was of rachitic origin.

In spite of the fact that immature infants have an increased susceptibility to and lessened resistance against infectious diseases during their first year of life, one finds that in the later years of childhood and adolescence they attain a degree of immunity that is surprising. Table 17 shows that these children do not suffer from any more diseases than the normal full term child, and in some cases even less. Especially striking is the great resistance that these children have against tuberculosis.

The Pirquet tuberculin test was made in 307 cases, and in only sixty of them, or in 19.5 per cent, was it positive. It is true that many of these patients were in the hospital during their first year of life, a period when the tuberculin test is rarely positive. We have, therefore, repeated the tuberculin test for all of our patients who came for reexamination. Four did not return to have the Pirquet test interpreted. In only thirty-four of the remaining ninety-eight cases, or in over one third of the cases, was the Pirquet test positive. When the results of the Pirquet test are arranged according to age groups and placed beside the

results of tuberculin tests made on normal children in Vienna, one is surprised to find that the tuberculin test of the immature child is less often positive than is that of the normal child.

In the sixty of the 307 cases in which the tuberculin test was positive, forty-one, or 13 per cent, actually showed roentgen-ray evidence of tuberculosis; in the majority there were only shadows of the hilum or the Ghon primary focus. Of these forty-one patients, four died of tuberculosis; twenty are alive and well, and the fate of seventeen is unknown. All the thirty-four patients who were reexamined and whose tuberculin tests proved to be positive were subjected to roentgen-ray examination of the lungs. In only fifteen of them was there a roentgenologically demonstrable pathologic process. In eight of them, there was tuberculosis of the hila of the glands; three of them showed shadows of the hilum with calcium infiltration; one had tuberculosis of the hila of the glands at the hilum and a Ghon primary focus; one had only the Ghon primary focus; two had tuberculous pleural effusions, one on the right and one on the left side.

Our material comes from the homes of poor working men, and the contention could not therefore be made that the small number of children with positive tuberculin tests is due to the fact that they come from better homes. Six of the children who gave negative reactions to the Pirquet test came from homes that were heavily laden with tuberculosis, and where one or more members of the family had already died from pulmonary or meningeal tuberculosis. It is true that the number of our cases is not large; yet they tend to show that if the immature children are not less susceptible, they are not more susceptible to tuberculosis than the full term child.

In a previous section the fact was mentioned that the most common type among our immature children, especially among the boys, was the asthenic type. The typus adenoidus or especially lymphatism was not unfrequently associated with this typus asthenicus. Over one half of our patients who came for reexamination complained of frequent catarrhal diseases of the rhinopharyngeal tract; some of these had a definitely adenoid facies. A considerable number had large pale tonsils. Especially striking was the great frequency with which enlarged cervical glands were found, especially the submaxillary and jugular groups. In a number of them there was a mild generalized lymphadenopathy with a barely palpable spleen. Unfortunately, blood studies were not made in these cases in order to determine also the presence or absence of a lymphocytosis. Some of these patients also looked anemic, but this was only apparent, as their mucous membranes were normally red. It would be interesting to follow these cases into later life, and to see how permanent this condition of lymphatism is and how these patients react to the diseases that are incident with adult life.

TABLE 17.—*Diseases Suffered by Immature Infants*

History Number	Grip	Icterus Catarrhalis	Pneumonia	Angina	Measles	Mumps	Scarlet Fever	Rubeola	Diphtheria	Otitis Media	Varicella	Bronchitis	Pertussis	Gastro-enteritis	Eczema
1533-1605	..	..	..	..	+	..	..	+	..	+	+	..	+	..	..
1750-1812	..	..	..	..	+	..	..	..	..	..	..	+	+	..	..
1521-1609	+	..	+	..	+	..	..	..	..	..	..	..	+	..	..
845-1650	..	..	..	..	+	..	..	..	..	..	+	..	+	..	..
1314-1705	..	..	..	+	+	..	..	..	..	..	+	..	+	..	..
1438-1535	..	+	+	..	..	..	..	..	..	..	..	..	+	..	..
1421-1533	..	..	..	..	..	..	..	..	+	..	..	..	+	..	..
942-1290	..	..	..	..	+	+	..	..	..	..	..	+	..	..	..
1638-1751	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
A-1416	..	..	..	+	+	..	..	..	..	..	..	+	+	+	..
1594-1570	..	..	..	+	+	..	..	..	..	..	..	+	+	..	..
2118-2042	..	..	+	..	..	..	..	..	+	+	+	..	..	..	..
1448- 681	+	..	+	..	..	..	..	..	..	..	..	..	+	..	..
1150- 823	+	..	+	..	..	..	..	..	..	..	..	..	+	..	..
1653- 80	..	..	..	+	..	..	..	..	..	..	+	..	+	..	..
688-1037	+	..	..	..	..	..	..	..	+	..	..	..	..	..	..
204- 185	+	..	+	..	+	+	..	..	+	..	..	..	..	..	+
W. Pr.	..	+	..	..	..	..	..	..	..	..	+	..	+	..	..
1576- 515	..	..	+	..	+	..	..	..	..	..	..	+	..	..	..
526- 528	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
1043-1180	..	..	+	..	..	..	..	..	..	..	..	..	..	..	..
753- 705	..	..	..	..	+	..	+	..	+	+	..	..	+	+	+
874- 900	..	..	..	..	+	..	..	..	..	..	..	..	+	..	..
777- 924	..	..	..	..	+	..	..	..	..	..	..	..	+	+	..
983-1006	..	+	..	+	+	..	+	..	+	..	..	..	+	..	..
842-1590	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..
1439- 439	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
450- 425	..	..	+	..	+	..	..	..	..	..	+	..	+	+	..
591- 660	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
283- 746	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..
791- 782	..	..	..	+	+	..	..	..	..	..	..	+	..	..	..
751- 803	..	..	..	..	..	..	+	..	..	..	..	+	..	..	..
740- 808	..	..	..	..	+	+	..	+	..	..	+	..	+	..	..
114- 811	..	..	..	..	+	..	..	..	..	..	+	..	+	..	..
925- 925	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
2124- 939	..	..	+	..	+	+	..	..	..	..	..	..	..	..	..
897-1028	..	..	..	..	..	..	+	..	..	..	..	..	..	..	..
1043-1057	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..
1125-1132	..	..	..	..	+	..	..	..	..	..	+	+	..	..	..
972-1191	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
1280-1293	..	..	..	..	+	..	..	..	+	..	..	+	+	..	..
528-1318	..	..	+	..	+	..	..	..	..	..	+	..	+	..	..
1333-1321	..	..	+	..	..	..	..	..	..	..	..	..	+	..	..
M. Za	..	..	..	..	+	..	..	..	+	..	+	..	..	..	..
1204-1332	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
1494-1522	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
1613-1621	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
1548-1723	..	..	..	..	..	..	..	..	..	..	+	..	+	..	..
1666- 825	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
A. Ha	..	..	..	..	..	..	..	..	..	+	..	..	+	..	..
1795-1826	..	..	..	..	+	..	..	..	+	..	+	+	..	..	..
C. F.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
160- 242	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
173- 302	..	..	+	..	..	..	..	..	..	..	..	..	+	..	..
318- 354	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
68- 332	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
816-2309	..	..	..	+	+	..	..	..	..	..	..	..	+	..	..
1256-1147	+	..	..	..	+	..	..	..	..	..	+	..	+	..	..
1252-1166	..	..	+	..	+	..	..	..	..	..	..	..	+	..	..
873- 789	..	..	..	+	+	..	..	..	..	..	..	..	..	+	..
1135- 986	+	..	..	+	..	+	..	..	..	+	..	+	..	..	..
1027- 921	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
31- 119	..	..	..	..	+	..	+	..	+	+	..	+	+	..	..
672- 370	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
280- 470	..	..	..	+	+	..	..	..	..	..	+	+	..	..	..
832- 287	..	..	+	+	+	..	..	..	..	..	..	..	..	..	..
1360- 10	+	..	..	..	+	..	..	..	..	..	+	..	..	..	..
30- 183	..	..	..	..	+	..	..	..	..	..	+	..	+	..	..
1639- 4	..	..	..	..	+	..	..	..	..	..	+	+	+	..	..
1517- 242	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
H-Ha	..	..	+	..	..	..	..	..	..	..	..	+	..	..	..
K-Ru	..	..	..	..	+	+	..	..	..	..	+	..	..	..	..
M-Lei	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..

TABLE 17.—*Diseases Suffered by Immature Infants—Continued*

History Number	Grip	Icterus Catarrhalis	Pneumonia	Angina	Measles	Mumps	Scarlet Fever	Rubeola	Diphtheria	Otitis Media	Varicella	Bronchitis	Pertussis	Gastro-enteritis	Eczema
632- 334	..	..	..	..	..	..	..	+	..	..	+	..	+	..	..
914- 35	+	..	..	..	+	..	..	..	..	..	..	..	+	..	..
1466	..	..	..	+	+	..	..	..	..	..	..	..	..	..	..
1561	..	..	..	..	..	..	..	..	..	..	+	..	..	..	..
894	..	..	..	..	+	..	..	..	..	..	..	..	..	..	..
354	..	..	+	..	..	..	..	..	..	..	..	+	+	..	..
505	..	..	..	..	..	..	..	..	..	..	..	+	..	..	..
779	..	..	..	..	+	..	..	..	..	..	..	+	+	..	..
1938	..	..	..	..	..	..	..	..	..	..	..	..	..	..	+
649	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
404	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
318- 354	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
68- 332	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
426- 449	+	+	..	..	+	+	..	..	..	..	+	..	..	..	..
408- 442	..	..	..	..	+	..	..	..	..	..	+	..	..	..	..
E. Sv	..	..	..	..	+	..	..	..	..	..	+	..	..	..	..
564- 564	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
673- 620	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
1782- 687	+	..	+	..	+	..	+	..	..	..	..	..	+	..	..
826- 910	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
95- 996	..	..	..	..	+	..	+	..	..	+	..	..	..	..	..
6- 740	..	..	+	..	..	..	..	+	+	..	+	..	+	..	..
847-1035	..	..	..	..	..	..	..	..	..	..	+	+	..	..	..
848-1056	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
1227-1485	..	..	..	..	+	..	+	..	..	..	+	..	+	..	..
1283-1361	..	..	..	..	+	..	..	..	..	..	..	..	..	+	..
1053-1229	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
948-1222	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..

TABLE 18.—*Tuberculin Reactions Compared*

Age	Tuberculin Test Positive	
	Monti's Figures for Viennese Children, Percentage	Our Immature Children, Percentage
First year	1	0
2 years	9	10
3 to 4 years	27	31.6
5 to 6 years	51	29.6
7 to 10 years	71	7
11 to 14 years	94	64

TABLE 19.—*Results of Pirquet Tuberculin Tests Made on Ninety-Eight Reexamined Immature Children*

	Positive	Negative
First six months	0	2
Six to twelve months	0	1
Second year	1	9
Third year	2	4
Fourth year	4	9
Fifth year	3	13
Sixth year	5	6
Seventh year	1	7
Eighth year	2	5
Ninth year	3	2
Tenth year	5	1
Eleventh year	4	0
Twelfth year	1	2
Thirteenth year	4	2
Fourteenth year	0	1

CONGENITAL MALFORMATIONS AND ANOMALIES

We encountered a variety of malformations among our immature infants which are worth reporting because of the unusual frequency with which they occurred in our material. Whether the malformations bear to the condition of immaturity the relation of post hoc or propter hoc, I am not prepared to say. It is conceivable, for instance, that a congenital cardiac defect, which leads to a subnormal blood supply to the organs, may be the cause of an infant of small birth weight. On the other hand, if the weight of an infant at the end of the ninth month of intra-uterine life was only that of one from six to seven months, it is also possible that an organ like the heart was affected by this general underdevelopment, with the resulting cardiac malformation.

There were ten cases, or 2.3 per cent, of congenital cardiac anomalies among the 437 cases of immature children. Seven of these came to postmortem examination; the other three are alive, and the only diagnosis entered in the history was congenital heart disease. Of the other seven cases, there was one case of simple foramen ovale, three of foramen ovale associated with patulous ductus botalli, one of aneurysm of the ductus botalli, one of pulmonary stenosis and one of dextrocardia with pulmonary stenosis. Doxiades also frequently found patulous foramen ovale in immature infants. He described the so-called fetal heart or condition of fetalism as characteristic for the heart of the immature child. This consists of a relatively small left ventricle, a large left auricle and a relatively large right side of the heart. The large left auricle is due to the patulous foramen ovale, which causes some of the blood in the right auricle destined for the pulmonary circulation to be shunted into the left auricle. It is of practical importance to bear in mind the frequency of congenital cardiac defects in immature infants, as the cyanosis caused thereby may sometimes be mistaken for cyanotic asphyxia due to intracranial hemorrhage, which is so frequent in infants of low birth weight.

Among the other defects found in immature children were Meckel's diverticulum in one case, clubfeet in one case, clubfeet and webbed fingers in one case, deformity of the toes in one case, congenital coxa vara in one case, cleft palate and harelip in one case and hemimicrosomatia in one case. Eight of the children had macrotia, unilateral or bilateral, usually the latter, and the ears were also abnormally projected forward. Thirteen of the children, or 3 per cent, had strabismus, seven unilateral and six bilateral, nine of them of the convergent and four of the divergent type. In two of the cases, the strabismus was associated with exophthalmos. There were five cases of pure exophthalmos alone. The oldest patient with strabismus was 19 years and 7 months old, and the youngest one was 4 years and 2½ months, the average age being 9 years and 1 month. Strabismus is frequently seen during the first two

months of life, also in many full term children; in the majority of the cases it then usually disappears. But in the immature child it more often persists into later life than in the full term child. The number of our cases of strabismus would have been greater if such a large number of immature children (over one-fourth) had not died during the first month of life. Ylppö said with reference to malformations in the immature infant:

> During the seventh and eighth month of intrauterine life, an extraordinary growth and change take place. We know that the body is most sensitive during periods of rapid growth, as during puberty, for instance. Hence, it is possible that during the period of transition from intra- to extrauterine life, that marked deviations from the normal occur.

OTHER DEVELOPMENTAL ANOMALIES

I have mentioned elsewhere that the types of anemias encountered among the older immature children are not types of true anemia but simply apparent anemia, perhaps due to vasomotor hyperactivity. During the first months of life, however, anemia is as common an accompaniment of immaturity as is rickets. It begins soon after birth, is definitely noticeable at the end of the first month and reaches its high mark between the fourth and sixth months. The anemia is usually the ordinary type of secondary anemia, but not infrequently there is also the chlorotic form, i. e., a relatively low hemoglobin content of the blood in comparison to the number of erythrocytes. After the sixth month, the anemia generally begins to disappear, but the repair is slow, so that even at the end of eighteen months one not infrequently encounters a marked anemia among immature children. The prognosis, however, is usually favorable, and the anemia has completely disappeared by the end of the second year.

According to Ylppö, spasmophilic diathesis or tetany is also common among immature infants. We have encountered it only six times among our cases, and it was of the manifest type; however, we are unable to say how many cases of latent tetany there have been among our large number of rachitic immature infants.

Owing to the small amount of subcutaneous fat in immature infants, weak musculature, patency of the inguinal canals and general underdevelopment of the tissues, there is a marked tendency on the part of these infants to develop umbilical as well as inguinal hernia. Ylppö found that the largest number occurs in those with the smallest birth weight, as shown in table 20.

We counted ten cases of hernia in our material, four inguinal and six umbilical. The oldest child with a hernia was 13 years of age and the youngest 5 weeks, the average age being about 4 years. Operation was performed in three of the cases of inguinal hernia; one patient was 2

years of age, one 1 year, and one 5 weeks; in the latter, the hernia was incarcerated. The relatively larger number of umbilical hernias is due to the coincident diastasis recti, which was present in eleven cases, all the cases of navel hernia being included among them.

The late persistence of the patency of the inguinal canals is the most important cause of the development of hernia in the immature children. In nine of our infants ranging from between 25½ months and 12 years of age, the inguinal canals were found to be widely open. In six of the cases, five bilateral and one unilateral, the testes were undescended, the oldest child being 10 years, and the youngest 1 year of age.

Among the 102 immature children who appeared for reexamination, there were four cases of hypophyseal disturbances. Three of them were cases of anterior hypopituitarism, all of the children being excessively adipose boys with an especially large amount of mammary and girdle fat and having small genitalia. The fourth one was a boy aged 12 years and three months, whose parents were of normal size and proportions,

TABLE 20.—*Ylppö's Figures on the Occurrence of Hernia*

Weight, Gm.	Number of Cases	Cases of Hernia	Hernia, Percentage
Up to 1,000	1	1	..
1,001 to 1,500	50	42	84
1,501 to 2,000	81	31	38.3
2,001 to 2,500	73	15	20.5

and who was 23.5 cm. too tall. He had strikingly large hands and feet, some prognathism, general hirsutism and external genitalia corresponding in size to those of a man of full sexual maturity. In placing these four cases on record, it is not my intention to link up the pathologic processes of hypophyseal disturbances with those of immaturity; nevertheless, I cannot resist the temptation to mention the following studies: Berberich examined forty pituitary glands of infants in whom Schwartz had found pial hemorrhages and thrombi, especially in the cavernous sinus and in the sinuses communicating therewith. In thirty-two of the forty glands examined, there was hyperemia due to the thrombosis, which in some of the cases led to atrophy of the rows of cells situated between the dilated capillaries. In four cases, there was actual necrosis (three times in the anterior and once in the posterior lobe). The chief seat of the pathologic processes was in the anterior lobe.

PROPHYLAXIS AND TREATMENT

After what has been said regarding the development of the immature child, it becomes evident that its future is not a promising one, and that it is wrought with many dangers. These dangers are greater the lighter the infant is at birth. Of the children weighing under 1,000 Gm. at birth, 83 per cent died before the end of the first year; of those weighing

between 2,000 and 2,500 Gm., only 30 per cent were dead at the end of the first year. When one considers that the child doubles its weight during the last two months of intra-uterine life, one realizes how rapid the daily growth is and of what fundamental importance each day of the last two months of intra-uterine life is for the development of the child. The obstetrician should, therefore, keep this fact in mind when he intends to induce labor prematurely. When the health of the mother is not in danger, every additional day that the child can be allowed to remain within the mother's womb increases its chances of vitality and healthy development. Hence, the later the induction of labor—if this is necessary—the better the immediate as well as the ultimate prognosis for the child. With the present low mortality rate in cases of cesarean section, this operation is desirable when premature labor is necessary because of disproportion between birth passages and passenger, so that the child may remain longer in utero.

When the child has been born, three facts must be kept in mind in order to insure a normal development, namely, prevention of infection, maintenance of body heat and proper nutrition. The first is carried out by strict isolation through exclusion of visitors, including all nurses who have rhinopharyngeal catarrh or any infection, and by asepsis in handling the child.

The maintenance of the body heat is important for the immature child, as it is a thermolabile infant. Subnormal temperature alone, by causing a too great cooling off of the body, is usually not a cause of death in infants, at least if it is not of too long duration. But this cooling off may lead to edema, scleredema and sclerema—the latter two conditions being simply a higher degree of the former—which immature infants show a great tendency to develop, especially smaller infants.

Thermoregulation in small and weak children has experienced a great many fads, chief among which were the large number of different forms of couveuses or incubators. It is now realized that a closed incubator is harmful because the humidity of the air is increased through the constant respiration of the child, and this moist air predisposes to broncho-pneumonia. The best ones are those couveuses in which the head of the child lies outside of the incubator while the rest of the body is warmed by electric bulbs or warm sand bags. Ylppö recommended that each infant be given a bath in water at a temperature of 39 C. after birth in order to prevent the initial cooling off; the child should then be placed in an incubator, and the longest time that it should be kept in there is from three to four weeks. Cook, in reporting on the importance of regulation of temperature for the premature infant, said that sixty infants were cared for by means of baskets with hot water bottles and seventeen by means of incubators. The mortality of the former amounted to 46.6 per cent, and of the latter to only 29 per cent.

Before going into the question as to what constitutes the proper nutrition for an immature infant, it is well to mention that it is of great importance to prevent the physiologic loss in these infants. The loss, of course, depends on the weight at birth; the heavier they are, the more they lose. Immature infants absolutely lose little, but relatively much. Hence, it is most essential to have this in mind and to so regulate the feeding from the start that the physiologic loss is at a minimum. Most of the smaller infants will at first have to be fed by gavage and fed frequently (from eight to ten times in twenty-four hours), because the capacity of the stomach is small.

As to what is the proper nutrition for an immature infant is debatable. Most pediatricians agree that when breast milk is available, it is the ideal food. It has been found, however, that the body of the immature infant requires more protein for rapid growth than is contained in breast milk, which was intended for full term infants. Some, therefore, advise the addition of from 1 to 2 per cent of protein to the expressed breast milk. Ylppö said that next to breast milk, simple milk dilutions, such as 50 per cent milk and 5 per cent cane sugar, are best. Others propose buttermilk to which carbohydrates have been added. In the clinic of one of the greatest experts on nutritional problems of children in Germany, milk to which 10 per cent of carbohydrates has been added is being used as the feeding mixture for immature infants. In brief, as in the feeding of full term infants, the methods are many and the success of all almost equally good, provided the caloric supply is sufficient (immature children have a very high energy quotient, requiring from between 150 to 175 calories per kilogram of body weight) and provided the children are kept free from infection.

SUMMARY AND CONCLUSIONS

Investigations into the fate of 437 children who weighed less than 2,500 Gm. at birth yielded the following results:

1. Two hundred and seventy-six, or over 72 per cent, were premature; 103, or 27 per cent, were born at full term, and two past term.

2. Over one half of the infants were first born, about one fourth second born, and about one eighth third born; in other words, the majority of the immature children of our series were first born children.

3. Two hundred and forty-four, or 56 per cent, were females and 193, or 44 per cent, males.

4. Syphilis plays a small part in the etiology of immature births, there having been among our cases only thirteen, or 3 per cent, in which syphilis was the cause.

5. The mortality of immature infants is high, especially among those whose birth weight is up to 1,000 Gm. Almost 40 per cent of those with

a birth weight under 1,000 Gm. die at the end of the first twenty-four hours and only 1.5 per cent of those weighing between 2,001 to 2,500 Gm.: in other words, the chances of survival are dependent first and foremost on the weight at birth.

6. Only 55 per cent of immature children are alive at the end of the first year and 52 per cent at the end of the fourteenth year of age. The critical period for the immature child, therefore, so far as survival is concerned, is the first year of life.

7. One of the most important causes of death of the immature infant during the first week of life is intracranial hemorrhage. This is due, not only to the direct mechanical trauma associated with the birth, but also to the vasolability of the immature infant. The vasolability is the result of two factors: (1) the persistence of the fetal state of the capillaries, i. e., the archicapillaries, and (2) the incomplete development of the vascular elastic tissue at the time of birth.

8. Of those that survive the first year, the largest number remain underweight and below the normal height, being more subnormal in the former respect than in the latter, even at the age of puberty. The subnormality in height is greater among the boys and that in weight, among the girls.

9. Based on calculations of the sitting heights of immature infants, we arrive at the conclusion that the chief cause of subnormal height lies in the delay of growth of the extremities rather than in that of the trunk.

10. The skull of the immature child, outside of the relative megacephaly during the second to the eighth months of life, is smaller than that of the mature child up to the twelfth year of age, especially during the first six years of life. After the twelfth year, the skull becomes normal in size and, in some cases, even overtakes that of the mature child.

11. The circumference of the chest also is smaller in the immature child up to the fifth year; after this it assumes larger proportions and continues to remain above normal until past puberty.

12. By far the larger number of immature children, especially boys, are of the asthenic and adenoid type, even though they are not of normal height. They are thin, have a long trunk and short extremities, a small or moderate deposit of subcutaneous fat, and are usually hypotonic. As the immature child forms a definite constitutional type, and since its height and weight have not become normal by puberty, it is possible that the retardation is not transitory, but will remain as a permanent condition of physical subnormality.

13. The static functions, sitting, standing and walking, begin later in the immature than in the full term child.

14. A large number of immature infants begin to talk much later than normal children, and defects in speech are more frequently encountered in the former group.

15. Only one third of the immature children that were of school age were in the classes that corresponded to their ages; twelve, or 27 per cent, were one year behind and nine, or 20 per cent, two years; two were three years behind and two were four years behind in school. Five children, or 11 per cent, of immature children of school age were attending schools for mentally inferior children.

16. Epilepsy (5 per cent), Little's disease (5 per cent), idiocy and imbecility (7 per cent) and enuresis (five cases) are far more frequent among immature children than among children whose birth weight was over 2,500 Gm.

17. Especially frequent among immature children is the condition of neuropathy, whether of the digestive, vasomotor or asthenic type.

18. There is no delay or retardation in the development of the signs of puberty among immature children.

19. During the first year of life, the immature child has little resistance to infections, and it is especially liable to infections of the respiratory tract.

20. Rickets and anemia are early accompaniments of immaturity for which the child should be treated prophylactically from the beginning of life, for the former by exposures to the quartz lamp or perhaps ergosterine, for the latter by large doses of inorganic iron.

21. Dentition is retarded in many cases as a result of the early presence of rickets.

22. After the first year of life, the immature child has a resistance to infections not unlike that of the mature child.

23. Tuberculosis is not more frequent among immature than among mature children; the tuberculin test is positive among the former less often than among the latter.

24. Congenital cardiac defects occurred more frequently among the immature children in our series than among the full term children.

25. The fate of immature children is not enviable; almost one half of them die during the first year of life. Of those that remain alive, the majority are physically as well as mentally underdeveloped. Some of them show a late mental development; others show a condition of psychic infantilism, if the term may be used in its nonspecific sense; while still others show permanent and severe mental diseases. When they are passed in review, one is surprised at the variety and amount of abnormality encountered, almost a regular "pathologic museum." In brief, the immature infant becomes the backward school child, and is a

potential psychopathic or neuropathic patient and even a potential candidate for the home for imbeciles and idiots. In view of the great frequency of immature births, the importance of this subject and also its practical significance to the mother, physician, school teacher, eugenist and even to the economist become evident. Efforts should therefore be directed not only to the care and preservation of the immature child, but more especially to the prenatal care and hygiene of the mother, prevention of maternal infection and correction of anomalies of the birth passages before pregnancy. Whenever possible, labor should not be induced prematurely, but the cesarean operation should be substituted. This will allow the child to remain longer in utero and to have attained sufficient weight and development when born to escape the pathologic conditions to which the immature child is predisposed.

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Pediatric Biographies

BARTHOLOMAEUS METLINGER
-1491

JOHN RUHRÄH, M.D.
BALTIMORE

The second pediatric incunabulum is a work of remarkable interest and one of the most vital of all the early texts on this subject, as is evidenced by the great number of times it has been reprinted. The first edition came from the press of Günther Zainer of Augsburg and is dated "Geschenen, als man zalt nach Christi Geburt tauset vierhundert und in den LXXIII Jar, an dem achten den Tag Sant Endris des zwölf Boten" (Dec. 7, 1473). It is entitled "Ein Regiment der jungen Kinder," and was written by a young physician of Augsburg, Bartholomaeus Metlinger.

His father's name, Dr. Peter Metlinger, according to Sudhoff, appears in the tax lists of Augsburg, and the son's name appears as "Meister Barthelme," (once as "Dr. Barthelme Mettlinger") regularly from 1472 until 1491, or the beginning of 1492. Besides the fact that he was inscribed in the Bavarian University of Ingolstadt, little is known concerning him.

His book, however, was reprinted again and again, in 1474, 1476, 1497 and 1500. It was reprinted twice in the sixteenth century, in 1511 and in 1531. It is difficult to trace the subsequent reprintings, as the text underwent changes at the hands of various printers and authors; but it appeared frequently along with Rösslin's "Rosegarten," a work on obstetrics, and in this way an English version by Jonas was printed. Metlinger's little book finally became more or less of a literary curiosity, until 1904, when Ludwig Unger reprinted the text in somewhat modernized German. In 1925, Sudhoff issued a facsimile of the first edition, and in the same year the author of this sketch included in "Pediatrics of the Past," a full English translation.

Metlinger used much the same sources for his material as Bagellardus, adding new material of his own, but the ancients and Arabians furnished a good share of it. He took his material, directly or indirectly, chiefly from Galen's first book, "De regimen sanitates," the canon of Avicenna, the Colliget of Averroës, the Pantegni of Constantine and the Continens of Rhazes.

Metlinger's first two chapters are writen on the care of the new-born, nursing, weaning and the choice of a wetnurse. If one reads this chapter carefully, one will find that people of the present day have not advanced so far as they think they have.

Glandular therapy is foreshadowed in the suggestion to feed the udder and bag of sheep and goats that have given milk; this, of course, is just as new as Soranus.

The third chapter or section deals with the various diseases seen in infancy and in childhood, beginning with those of the head and progressing downward. It includes articles on: meningitis, hydrocephalus, sleeplessness, cramps, paralysis, running ears, inflammation of the eyes, swelling of the neck, diseases of the mouth, bronchitis, diseases of digestion, jaundice, dysentery, constipation, worms, abdominal pain, umbilical and inguinal hernia, stone, ulcers of the skin, fever, erysipelas, measles and smallpox.

The fourth chapter considers the child from the time it learns to walk up to the seventh year: its physical and moral upbringing, eating, drinking, bathing and exercise. The education is to begin at 6 years of age. Muss (Brei), or mush and milk, is the chief diet during the early years, and other articles are added as the child grows. Wine is not given to healthy children under 7; it may be given to girls at 12 years of age and to boys at 14.

While the whole text is well worth reading, it is impossible to give but one short extract, the section on the management of the new-born and the choosing of a wetnurse. The woodcut is interesting, as it is one of the first pediatric illustrations. It shows a charming fifteenth century interior with the maidens spinning, the boy reading and the baby swaddled in its crib.

In the first fourteen days it is better that another woman suckle the child as the milk of the mother is not as healthy, and during this time the mother should have her breasts sucked by a young wolf or the milk should be sucked off. When, however, the mother will nurse the child from the first day then one should put a small amount of honey or rose honey in the mouth so that the milk injures it less. And now if one will have a child nursed the wet-nurse should first milk the breasts so that the watery part runs from it and then give the child to suck. It must be considered that if the child is to be healthfully nourished both wet-nurse and the child should be well ordered and live properly because where this is not done all sorts of diseased conditions may arise. The best wet-nurse for a healthy child is its own mother but in sickness or where the milk does not come in or for other reasons the mother cannot nurse her child, one should choose a wet-nurse who has the following appearance and habits. She must neither be too young nor too old. She should be not younger than twenty years of age and at twenty-five she is at her best. Her own child should be over six weeks old. It is best when she has suckled two or three other children. The

time of her delivery should not have been too soon either with this child or with the others. She should be well built; her face healthy in appearance, tanned; and she should have a strong thick neck, strong broad breasts, not too fat and not too thin but preferably well formed and fleshy and that do not hang down, not too small but average good size. The wet-nurse should have good praiseworthy habits. She should not be easily frightened or worried and not small-minded or prone to anger. She should be industrious and careful with the child. She should take care that she does not become pregnant as this is dangerous to the child, as the best blood of the pregnant woman goes to the fetus. In such cases the child should be weaned just as if the wet-nurse herself were sick. The less the child nurses at this time the better it is for the child. Also the wet-nurse should not be diseased.

News and Comment

GIFT TO CHILDREN'S HOSPITAL OF CINCINNATI

Mr. William Cooper Procter, the president of the Board of Trustees of the Children's Hospital of Cincinnati, has announced the gift of $2,500,000 to that institution to be expended mainly for teaching and investigation. It is planned that approximately $500,000 will be spent in the development of a building, housing research laboratories and the necessary adjuncts thereto, and in further development of the outpatient clinic. The Children's Hospital of Cincinnati is closely affiliated with the College of Medicine of the University of Cincinnati through its pediatric department — the professor of pediatrics in the College of Medicine being the chief of staff and medical director of the Children's Hospital. The Children's Hospital has been pronounced by all those who have seen it as the finest institution of its type which has yet been erected. The fact that this building is entirely paid for and already has heavy endowment allows for the finest type of care for patients and also permits the expenditure of the sum previously mentioned for purposes not directly concerned with the care of patients. This gift of Mr. Procter's is of special significance in that it not only permits a tremendous expansion of the type of work for children already in existence in Cincinnati but that it is to be largely employed in the pursuance of investigation along several lines such as clinical, laboratory, preventive and social service work. The pediatric department does not confine its activities to the care of patients and the teaching of undergraduate and graduate medical students but, through its affiliation with national and local organizations, is in close touch with preventive pediatric work and problems connected with the health of the city. Under its direction or in association with it are other hospitals and institutions concerned in the care of children and in the prevention of disease. The affiliations are such that it is possible to give undergraduate and graduate instruction in all types of pediatric work. Among its most important connections is that with the Babies' Milk Fund Association, of which B. K. Bachford is medical director. The policy of the pediatric department has been and will be the coordination of voluntary, part-time and full-time staff in teaching, investigation and care of patients.

DECREASE IN BIRTH RATE

The United States Public Health Service notes in its latest bulletin that the birth rate in the registration area decreased from 21.1 births per 1,000 population during the calendar year 1925 to 20.1 in 1926. This is a continuation of the trend which has been noted since 1915, when the birth registration area was established. In that year the birth rate was 25.1 per 1,000. The phenomenon of decreasing birth and death rates is not peculiar to the United States; it has been noted in most civilized countries.

The infant death rate is an important index of the efficiency of public health work. In the United States this rate has been decreasing for several years, although in 1926 there was a slight increase. Reports from twenty-eight states give the rate of 72.8 deaths of infants under 1 year of age per 1,000 births in 1926, while the rate for 1925, in the same states was 71.5 per 1,000 births.

CONFERENCE ON RHEUMATIC DISEASES

A conference on rheumatic diseases is to be held at Bath, England, on Thursday and Friday, May 10 and 11, 1928. Sir George Newman, Chief Medical Officer of the British Ministry of Health, has consented to act as president of the conference. There will be three sessions. The subject for

discussion at the first session will be "Social Aspects," and Lord Dawson of Penn, Physician to H. M. King George will preside; the subject for discussion at the second session will be "Causation," and Sir Humphry Rolleston, Regius Professor of Physics, University of Cambridge, will preside. The subject for the third session will be "Treatment," and Sir E. Farquhar Buzzard, Regius Professor of Medicine, University of Oxford will preside. The local Hon. Medical Secretary is Dr. Vincent Coates, 10, Circus, Bath, England.

EXPENDITURES OF COMMONWEALTH FUND

Last year, $1,100,000 was expended by the Commonwealth Fund in efforts to improve the physical and mental health of American children. Other gifts for hospitals and for educational and welfare work brought the total appropriations for the fiscal year ending Sept. 30, 1927, to $1,953,557.

The capital endowment of the Commonwealth Fund, which was established in 1918 as a general philanthropic foundation with an initial gift of $10,000,000 from the late Mrs. Stephen V. Harkness, was increased by additional donations during her lifetime and now amounts to more than $38,000,000. The income last year was $2,129,748.

Approximately $417,000 was expended to carry on the Fund's program for the promotion of child health.

Abstracts from Current Literature

Pathology, Bacteriology and Biochemistry

Transmissible Toxicogenicity of Streptococci. Martin Frobisher, Jr., and J. Howard Brown, Bull. Johns Hopkins Hosp. **41**:167 (Sept.) 1927.

"Nontoxicogenic, nonscarlatinal streptococci and strains of *B. coli, B. prodigiosus, B. subtilis* and *S. albus* have been cultivated in mixed culture with toxicogenic, scarlatinal streptococci and then recovered in pure culture. Two nonscarlatinal streptococci were found to have acquired, temporarily, a definite toxicogenicity, giving rise to a toxin which is neutralized by antiscarlatinal serum. The other organisms did not acquire any toxicogenicity.

"Non-toxicogenic streptococci may acquire a toxicogenicity from sterile Berkefeld filtrates of the scarlatinal types, in general, less marked than those acquired from the mixed cultures. The existence of a filtrable second factor associated with the scarlatinal streptococci is suggested."

Authors' Summary.

Coccal Forms of the Diphtheria Bacillus. H. J. Parish, Brit. J. Exper. Path. **8**:162, 1927.

From time to time coccal forms may appear in tryptic digest-broth cultures of *Bacillus diphtheriae*. These coccal forms are virulent and toxicogenic and do not appear to be in any sense degenerative.

The Botelho Reaction in the Diagnosis of Cancer. B. Bisbini, Clin. Pediat. **9**:387 (April) 1927.

The Botelho reaction is intended to demonstrate the presence of specific substances apparently produced by neoplastic tissue in the blood serum of patients with cancer. It is based on the property of human serum to give a precipitate when in contact with a mixture made up of an iodine solution and citric acid. This property is more pronounced in cancerous serum.

The preparations used are made up as follows:

a. Citric acid	0.5	Gm.
Water	100	cc.
Solution of formaldehyde	1	cc.
b. Metallic iodine	0.1	Gm.
Potassium iodide	0.2	Gm.
Water	210	cc.
c. Physiologic solution of sodium chloride		

The blood obtained when the patient is fasting is allowed to stand in a cool place for twenty-four hours. If the serum is not clear at the end of this time, centrifugalization is necessary. The serum is then diluted with equal parts of physiologic sodium chloride solution. Of the diluted serum, 0.5 cc. is then mixed with 2 cc. of the citric acid solution and agitated. With the addition of 0.7 cc. of the iodine solution, a cloudiness appears which disappears if the serum is noncancerous. If 0.2 cc. more of the iodine solution is added, a precipitate forms which will disappear on shaking if the serum under examination is not cancerous. The precipitate will remain for twenty-four hours or more if allowed to stand. Sometimes a deposit accumulates at the bottom of the tube and the supernatant fluid becomes clear. Mere persistent cloudiness and formation of a heavy precipitate denote gradations in the positivity of the reaction.

The Botelho reaction is advocated because:

1. It offers a large percentage of positive results. In a series of seventy-five patients suffering from various diseases, exclusive of cancer, only three gave a positive reaction. Of ten with cancer, seven gave a positive response.
2. The technic is simple.
3. It may serve as a valuable adjunct to other methods of investigation.

ZAMBARANO, Northville, Mich.

NORMAL BILIRUBIN CONTENT OF SERUM. R. SIVO, Biochem. Ztschr. **189**:159 (Oct.) 1927.

The author employs a new method for determinations of bilirubin. The "indirect" bilirubin is freed from its combination with protein by the addition of caffein, which gives a more accurate determination after precipitation of the protein. In 200 patients who were either well or who did not have anemia, infectious diseases or disease of the liver, the following results expressed in milligrams (percentage) were obtained: 82 per cent between 0.80 and 1.10; 9.5 per cent between 0.65 and 0.80; 8.5 per cent between 1.10 and 1.20. None of the many cases showing pathologic changes presented less than 0.65 mg. per cent. A value in the upper region of normal may be pathologic for a particular person.

DECOMPOSITION OF BILIRUBIN SOLUTIONS. R. SIVO and E. FORRAI, Biochem. Ztschr. **189**:168 (Oct.) 1927.

Crystalloid preparations of bilirubin and urine containing bilirubin decomposed rapidly in the sunshine, and within twenty-four hours when kept in the dark. In serum or in other liquid containing colloids, the decomposition was slight, as determined by the iodine reaction. It is important to have a fresh specimen of urine when testing for bilirubin.

BRAHDY, New York.

THE VITALITY OF SCARLATINAL STREPTOCOCCI. H. M. VON JETTMAR, Ztschr. f. Hyg. u. Infektionskr. **107**:265, 1927.

The author found hemolytic streptococci in 100 per cent of 120 cases of scarlatina. Pure cultures of them or mucus from the throat containing them were dried on cotton and kept in the dark at room temperature. Living streptococci could be obtained from this material for over one-half year. The floating dust scraped off from the pieces of cotton contained living, fully virulent streptococci. Of the streptococcal flora from the mouth of patients with scarlet fever the hemolytic streptococci survive the drying longest, while those in the viridans group die within a few weeks. Hemolytic streptococci which have been in a dried condition for a long time at first show a retardation of growth on artificial culture medium which disappears rapidly. When tested on animals, they prove to be fully virulent. Their production of toxin also remains unaltered. Even exposure to direct sunlight and to other atmospheric influences for days does not destroy them. The results of the experiments demonstrate the great resistance of the scarlatinal streptococci and the possibility of infection by dry dust.

W. OPHÜLS.

Nutrition

THE CHEMISTRY FINDINGS IN RICKETS. JAMES L. GAMBLE, Boston M. & S. J. **197**:373 (Sept. 8) 1927.

This is the first of three papers of a symposium on rickets presented before the 1927 meeting of the Massachusetts Medical Society. No original work is presented in this paper, but the author discusses in an interesting and helpful manner the work done by Howland and Kramer, Hess and Steenbock

and Park, Guy and Powers. The former showed that ordinary rickets is seen when there is low blood plasma phosphorus even with normal blood plasma calcium, while tetany occurs with low blood plasma calcium even with normal blood plasma phosphorus. They also showed that there is a fairly close correlation between the size of the Ca × P products (normally between 50 and 66) and the degree of roentgen-ray and clinical rickets. The fact that the amount of this product is usually below normal in patients with tetany explains the presence of the associated rickets.

Hess and Steenbock showed that rickets also occurs when there is a deficiency of an organic substance known as vitamin D which is found in cod liver oil and which may be produced by exposure to the ultraviolet rays of the sun or quartz lamp; they also showed that by irradiation many substances could be made antirachitic. Park, Guy and Powers showed a rough correlation between the values found for calcium and phosphorus in the blood plasma and the amounts of these substances in the diets; they also showed that when these substances were deficient in the diet the administration of cod liver oil with its high content of vitamin D brings about a rapid restoration of the normal values for plasma calcium and phosphorus.

THE PREVENTION AND TREATMENT OF RICKETS. EDWIN T. WYMAN, Boston M. & S. J. **197**:376 (Sept. 8) 1927.

This paper is especially interesting since it reports the results of treatment of rachitic infants by exposure to sunlight coming through several types of window glass which had previously been tested at the Massachusetts Institute of Technology and found to transmit the ultraviolet rays of the sun.

Four patients were treated with the sun rays through quartz glass, three through Corning glass, and two through Vitaglass. Three infants with severe rickets treated last year in front of the quartz window made rapid recovery; however, as it was not definitely known how much antirachitic treatment, if any, they had received previous to admission to the Children's Hospital, all of the infants in this study were kept in the hospital for from ten to twenty days before the sunlight treatment was started. All of the infants except the two treated through the Vitaglass showed definite evidence of recovery as determined by clinical evidence, roentgenograms and examination of the blood. The author suggests that while the Vitaglass did not transmit sufficient ultraviolet rays to be of use in treating the two negro infants during the winter months in Boston, it is possible that if the windows had been so situated as to give longer exposure to the sun's rays, the results would have been different.

A PLEA FOR SANITY IN THE USE OF THE MODERN METHODS FOR THE PREVENTION AND TREATMENT OF RICKETS. JOHN LOVETT MORSE, Boston M. & S. J. **197**:388 (Sept. 8) 1927.

Dr. Morse suggests that many physicians overlook the fact "that rickets is no more common now than in the past, that it is less severe than it used to be, and that much of what is called rickets is probably not really rickets at all, but a physiologic change, and certainly not of importance." He asks, "Is it advisable to give all babies cod liver oil from the time they are born, regardless of how they are fed, their social status, how much sunlight they get and the time of the year"? He also questions the necessity of using artificial ultraviolet irradiation, not only in all cases of rickets but in many other conditions, as is being done by so many physicians. He says: "It is apparently taken for granted that it is impossible to do any harm with artificial ultraviolet irradiation, even if it does no good. There is ample experimental evidence to show that the prolonged irradiation of cholesterol and of cod liver oil and of foods which have acquired antirachitic powers, as the result of irradiation, diminishes and finally destroys these antirachitic powers. Furthermore, when cholesterol has been inactivated by artificial irradiation, it

cannot be reactivated. It is evident, therefore, that too prolonged irradiation of babies may, instead of activating the cholesterol in the skin, not only inactivate it, but prevent it from being activated by sunlight." He also calls attention to the possible beneficial action of the red and infrared rays, as well as the ultraviolet rays.

GENGENBACH, Denver.

THE BLOOD PEPTIDE NITROGEN IN ARTERIAL HYPERTENSION. H. JACKSON, JR., D. W. SHERWOOD and O. J. MOORE, J. Biol. Chem. **74**:231, 1927.

The blood peptide nitrogen does not rise sufficiently in hypertension to be of etiologic importance.

EFFECT OF ANTIRACHITIC VITAMIN ON THE PHOSPHORUS, CALCIUM AND p_H IN THE INTESTINAL TRACT. L. YODER, J. Biol. Chem. **74**:321, 1927.

Both irradiation and the administration of cod liver oil produce a lowering of the p_H in the intestinal tract of rats on a rachitic ration. This decrease in p_H may be responsible for the associated increased utilization of calcium and phosphorus.

ARTHUR LOCKE.

THE ACID BASE BALANCE IN CHILDREN WITH REFERENCE TO INTESTINAL DISTURBANCES. E. B. WOODS, J. Missouri M. A. **25**:1, 1928.

While neither original nor new facts are brought out, the article is of great value to the pediatrician for a summary and review.

SCHORER, Kansas City, Mo.

THE PUBLIC HEALTH VALUE OF CONCENTRATED MILK. JAMES A. TOBY, M. J. & Record **126**:208 (Aug. 17) 1927.

Milk is the most valuable of foods, but it may be one of the most dangerous, due to the many possibilities of contamination. The desirability of some method for preserving whole milk was recognized more than a century ago. The forms and the several processes of preparing concentrated milks have a wider scope of usefulness from the point of view of public health, because of the following reasons: (*a*) They are clean and safe; (*b*) they may be kept for long periods of time; (*c*) they are uniform in composition; (*d*) they are more digestible, and they are convenient and inexpensive. Their nutritional qualities are not impaired by the manufacturing process. The vitamins are present in the concentrated milks in approximately the same amounts in which they were present in the milk before the process began. The concentrated milks may be used for all of the same purposes as pure whole milk.

CLINICAL OBSERVATIONS ON COD LIVER OIL CONCENTRATE IN RICKETS AND OTHER NUTRITIVE DISTURBANCES. JACOB COHEN, M. J. & Record **126**:308 (Sept. 7) 1927.

The author advocates the use of oscodal, a concentrate prepared from cod liver oil said to contain the antirachitic and the antiophthalmic vitamins in all cases in which cod liver oil is called for. It is more palatable and children take it well.

Four case reports are given in the paper showing the efficiency of this preparation.

WILLIAMSON, New Orleans.

INFANT FEEDING IN RELATION TO DISEASES AT ALL AGES. A. A. SHAWKEY, W. Virginia M. J. **23**:398 (Aug.) 1927.

Improper feeding in infancy and early childhood is discussed as a direct or indirect cause of all diseases at all ages. Proper and improper feeding are discussed briefly. Prominent authorities are quoted on various phases of the

subject. Case histories are abstracted briefly, and a survey is given of 1,000 case histories taken at random, showing that more than 76 per cent of the illnesses were caused wholly or in part by errors in feeding.

The author believes that the influence of feeding in infancy and early childhood on the health, efficiency and longevity of the individual is great; that the subject is one that is most important and little understood, and that it merits a great deal of study and investigation.

AUTHOR'S SUMMARY.

TREATMENT FOR RICKETS. A. B. MARFAN, Paris méd. **45**:358 (Nov. 5) 1927.

The treatment for rickets is divided into two parts: first, medication against the cause, and second, medications that favor general nutrition, and more especially the bones. The specific treatment consists of fresh air, cutaneous stimulation and systematic heliotherapy. The medicines used are limited to cod liver oil, calcium preparations, epinephrine and in certain cases, phosphorus.

It is remarkable how many badly deformed bones will return to normal under systematic treatment. However, if results are not conclusive after 4 years of age, orthopedic appliances are recommended for the limbs. Thoracic deformities are best treated by corrective gymnastics.

PERSISTENT VOMITING IN INFANTS. G. VARIOT, Progres méd. **44**:1694 (Oct. 29) 1927.

In persistent vomiting of breast-fed infants due to a high fat content of the breast milk, the author has had good results with dried milk low in fat. He believes that this may be better than attempting to keep the child at breast. He also believes that the differences in opinion between the value of condensed milk, existing in France and the United States, are due to differences in standardization.

SANFORD, Chicago.

PREDISPOSITION AND NUTRITION. TADASEA SAIKI, Ann. de l'Inst. Pasteur **41**:668, 1927.

Guinea-pigs and rats fed on diets deficient in vitamins A, B and C are more resistant to strychnine than are normal animals. A regimen deficient in vitamin A and rich in calcium phosphorus produces stones of the bladder and kidney and gallstones in rats. By means of roentgen-ray examinations cures were noted in six rats with stones after they were fed a diet rich in vitamin A. Rats kept alive over long periods of time with a minimum vitamin A diet were found to develop typical squamous cell carcinoma of the stomach and bladder with lesser changes in other organs, one rat showing metastasis of the lung. Local irritation was not used and evidence of ulcers was not found. In relation to the formation of cancerous growth the cicatrization of wounds was noted under various vitamin regimens, and it was found that a diet deficient in vitamin A favored healing or proliferation of epithelial tissue.

G. B. RHODES.

THE PROCEDURE OF DE BUYS AS A PROPHYLACTIC TREATMENT AGAINST SOME DISORDERS OF BREAST FEEDING. LEON VELASCO BLANCO and HUMBERTO PAPERINI, Arch. Americanos de med. **3**:99, 1927.

The authors report favorable results with the treatment of the breast of nursing mothers with the strapping procedure of De Buys. It is an excellent preventive measure against the development of abscess of the breast.

The technic is simple. The strapping is well borne and supports the breasts far better than suspensories. In not a single instance was diminution of secretion observed. On the contrary, the capacity of the mother to nurse was immeasurably increased. The application permits the treatment of any disorder of the nipples.

SCHLUTZ, Minneapolis.

CHEMICAL COMPOSITION OF BONES IN SCURVY. L. BROUWER, Biochem. Ztschr. **190**:402 (Nov. 23) 1927.

Acute scurvy was produced in guinea-pigs by feeding them a scorbutic diet for several weeks. The amount of ash, calcium and phosphorus in the bones was 10 per cent less than in carefully selected normal controls. The Ca: P ratio was not changed. The changes are less than the author expected to find.

BRAHDY, New York.

FEEDING OF INFANTS. L. LANGSTEIN, Gesundheit. f. d. Kindesalter **1**:420 (May) 1926.

Langstein criticizes the complicated and varied types of feeding now in use, and believes that the simple milk dilutions with at least 6 per cent carbohydrates are sufficient for the normal infant in a proper environment. He regards the latter as essential to the sucess of the feeding. He advises that the public be taught in simple words the essential facts of infant care and that the general practitioner be not confused with complicated schemes of feeding.

REICHLE, Cleveland.

THE INFLUENCE OF COOKING ON THE NUTRITION OF FOOD. FRITZ VON GRAEVENITS, Monatschr. f. Kinderh. **37**:36, 1927.

Previous investigators have reported experiments in rats showing that the curves of growth were more satisfactory when raw food was used than when the food had been cooked. The author repeated these experiments with particular emphasis on the use of raw and cooked eggs. He could not confirm the previous investigations and believes that the cooking made little difference in the nutrition of the food.

GERSTLEY, Chicago.

DETERMINATION OF GASTRIC FUNCTION IN INFANTS. J. MOSONYI and I. LEVAY, Orvosi hetil. **71**:520, 1927.

Chloride content of the blood was determined in infants before and during digestion. Infants who had a good appetite and who were gaining weight showed a decrease of chloride of from 0.03 to 0.04 per cent during digestion. Infants with poor appetite did not show any or only an insignificant increase in a few instances and in several cases an increase of the chloride content.

ZENTAY, St. Louis.

Prenatal Conditions

RARE MALFORMATION OF KIDNEY. F. DE GAETANI, Arch. per le sc. med. **49**:138, 1927.

The anomaly was found in an infant, aged 41 days, who died of capillary bronchitis. The author believes that the lateral torsion, especially of the left kidney, was greater in his case than in any other described before. The lower part of the hilum was turned laterally. Besides this, both kidneys were hypertrophic, lobated and slightly ectopic and the renal blood vessels anomalous.

K. SCHULHOF.

DICEPHALY. G. B. GRUBER and H. EYMER, Beitr. z. path. Anat. u. z. allg. Pathol. **77**:240, 1927.

Gruber and Eymer record an anatomic study of six examples of dicephaly with varying degrees of reduplication of the vertebral column. The degree of reduplication of the internal organs depends on the extent of duplication of the vertebral column. Heubner had claimed that every dicephalic monster has a double respiratory system; this was not true of the first case in the

present series. In one case with doubling only of the cervical vertebral column, four phrenic nerves had been formed. The position and shape of the internal organs is influenced by the space available for them during the developmental period. The contribution is embellished by a reproduction of an illustration of an adult dicephalic Turkish soldier, dated 1697, and that of a dicephalic child, dated 1512.

O. T. Schultz.

Free Transplantation of the Upper Extremity Through Amniogenic Malformation. L. Hirsch and H. Reineck, Frankfurt. Ztschr. f. Path. **35**:48, 1927.

The authors cite a case of extreme malformation in a new-born child in which there had been a transplantation of the left upper extremity to the lower part of the left trunk. They believe that an anomaly of the amnion effected a free transplantation of the "anlage" of the extremity. As proof of this they call attention to a demonstrable scar at the shoulder at the usual location of the arm and also to the peculiar innervation of the limb. Instead of segments C4 to D1 of the plexus brachialis supplying the part, the nerves are derived from segments D9 and 10. Furthermore, the scar in the skin on the left shoulder convexity shows visible patterns which correspond closely to the bone-building part on the head of the misplaced humerus. For these reasons the authors consider that the arm was originally formed in the normal place.

E. M. Hall.

Congenital Attitude Anomalies Caused by Muscular Contractions: Clubhand, Wry Neck and Knee Contractures. A. Hottinger, Jahrb. f. Kinderh. **112**:258 (May) 1926.

Three cases of congenital myogenic contractures are described by Hottinger. The first was that of a boy, aged 6 weeks. His mother's pregnancy had been normal, as had the delivery. The liquor amnii conditions had also been normal. The child, however, presented multiple deformities; his shoulders were held high, he had clubhand with osseous alterations, clubfoot, stridor, struma, umbilical hernia and cryptorchismus. The hands and feet became normal after six months of intermittent treatment with reduction bandages and massage. The second case was that of a girl, aged 5 weeks, with congenital wry neck. There was simple muscular shortening without hematoma. Her mother's pregnancy and delivery had been normal. Three months of posture and massage cured the anomaly. The third case was that of a girl, aged 5 years, with congenital contracture of the knees. The birth had been a breech presentation, with the knees prolapsed. She developed into a habitual knee walker. There was secondary atrophy of the legs and feet. The vastus was absent and the patella abnormally small. Normal function of the extremities was obtained in six months through reduction, massage and tenotomies. Infection, trauma, spina bifida and primary changes of the central nervous system could be excluded in all three cases. The second case was an anomaly of attitude resulting from uterine constriction. The other two may have been due to primary, perhaps familial, defective germ or habitual attitude anomalies due to a constrained position while in utero.

Pace, Chicago.

Congenital Encapsulation of the Small Intestine. R. Füth, München. med. Wchnschr. **74**:319, 1927.

A girl, aged 15, had intestinal obstruction with intermittent attacks of acute pain and a constant abdominal mass. The diagnosis made was ovarian cyst on a twisted pedicle. Operatively, a large encapsulated mass was removed which proved to be a peritoneal sac containing more than three fourths of the closely coiled small intestine which consisted only of mucus and muscular coats.

J. D. Willems.

CONGENITAL DEFECT OF THE DIAPHRAGM. P. CARTELLIERI, Virchows Arch. f. path. Anat. **263**:599, 1927.

Nine examples of defect of the diaphragm in fetuses and new-born infants are described in detail. In seven, the diaphragm was defective on the left side, in one on the right side, and in one there was left sided herniation without defect. In two cases, absence of the greater portion of the left side of the diaphragm was associated with herniation of the right side. Although Beneke's conception that excessive growth energy of the abdominal organs as compared with those of the thorax is a factor in the genesis of diaphragmatic defect is accepted as plausible, the author does not believe that his cases offer evident support of this theory. Jahn's theory that the primary condition may be a disturbance of growth of the pleuroperitoneal fold is held to be more probable.

BILOCULAR URINARY BLADDER. R. CHWALLA, Virchows Arch. f. path. Anat. **263**:632, 1927.

In a human fetus 32.5 mm. long, at which stage the ureteral membrane should have disappeared, the membrane had persisted on one side and by transposition toward the midline had caused the formation of an accessory bladder cavity.

IDENTIFICATION CHARACTERS OF SINGLE OVUM TWINS. H. W. SIEMENS, Virchows Arch. f. path. Anat. **263**:666, 1927.

Siemens has devoted much attention to the pathology of twins. He does not believe that the fetal membranes give an absolutely certain clue as to the single ovum origin of twins, nor that the finger prints are of any value in determining such origin of any pair of twins. The condition of the fetal membranes is not of help after birth, since the individual usually has no knowledge of this point. In the study of geminal pathology, it is important to know in any given case whether the twins have come from a single ovum or from two. Siemens maintains that the origin of any given pair of twins from a single ovum can be deduced by comparison of a sufficiently large number of external characteristics, of which he has selected twelve. These are as follows: form and color of the hair; color of the eyes and skin; distribution of freckles; distribution of congenital anomalies of the skin vessels, such as telangiectasis, cutis marmorata and acro-asphyxia; keratoses and folliculitis; grooves of the tongue and tooth form; face and head form; ear form; hands and nails; body form. To the so-called dermatologic identification characteristics, which he had previously used, he now adds three others which should be taken into consideration in any study of twins. These are psychic characteristics, congenital anomalies and diseases, and those characters which require for their detection more complicated methods, such as dactyloscopy, capillary microscopy and eye refraction.

CHONDRODYSTROPHIA FETALIS. A. KAHLSTORF, Virchows Arch. f. path. Anat. **265**:84, 1927.

In eight chondrodystrophic fetuses Kahlstorf found a smaller number of Hassall bodies in the thymus than in normal infants of the same ages, and eosinophilous cells were absent or greatly decreased in number. These changes are interpreted as evidence that chondrodystrophia is due to hypofunction of the thymus. The ingrowing periosteal streak, which separates the cartilage of the epiphysis from the diaphysis and results in failure of bone growth, is probably the result of persistence and hypertrophy of connective tissue which accompanies the vessels of the marrow canals of the cartilage.

O. T. SCHULTZ.

Diseases of the New-Born

A Study of Erythrocyte Diameters in the New-Born. Herbert Silvette, J. Lab. & Clin. Med. **13**:245 (Dec.) 1927.

Six thousand erythrocytes of thirty normal new-born infants were measured. The greatest number had a diameter between 7.2 and 8.0 microns. The average mean diameter was 7.45 microns, with an average variability of 4.88 microns.

Gibson, Elkhart, Ind.

Variations of the Leukocytes in the New-Born After Eating. G. Roi, Clin. Pediat. **9**:571 (July) 1927.

After the first ingestion of food, numerical changes are not observed in the leukocytes in new-born infants. Leukocytic variations are observed about the second day after alimentation is started. In debilitated and premature infants, this period is longer. The postalimentary changes noted are manifested in the majority of cases by a leukopenia. Hydrochloric acid could not be demonstrated in the gastric juice obtained after feeding. Histamine injected into fasting infants provokes a marked secretion of hydrochloric acid with a corresponding leukocytosis in the peripheral blood.

Zambarano, Northville, Mich.

Icterus Neonatorum of Calves. E. Metzger, Virchows Arch. f. path. Anat. **263**:703, 1927.

Icterus was present in 504 of 4,000 calves examined by the author. In 322 new-born calves included in the whole series, icterus was detected in twenty-four and the latter group was subjected to microscopic study. The author claims that in these animals the liver was not fully developed, a condition which manifested itself by irregularity of the liver cords and of the bile canaliculi. Metzger concludes that the icteric state is due to the persistence of the fetal condition of the liver, which permits bile pigment to enter the blood.

O. T. Schultz.

Intracranial Injury in the New-Born. Hans Heidler, Wien. klin. Wchschr. **40**:1408 (Nov. 10) 1927.

Heidler reviews recent investigations on intracranial injuries at birth. Mortality statistics on this subject to date have been inaccurate, due chiefly to lack of confirmation at autopsy. The author compared all of his clinical cases with those of Jaschke. He found that 0.82 per cent of all of the patients died from intracranial injuries; Jaschke's figure was 0.22 per cent. Martin found intracranial injury to be sixteen times more frequent in artificial than in natural deliveries, while Heidler, in a comparative study of his cases during 1925, showed the incidence to be less than twice as great. In the latter's series (2,827 births) the mortality from this cause was 1.9 per cent, after small premature infants, deaths from destructive operations, etc., were excluded. Fifty per cent of all deaths were due to injuries to the head at birth.

Although Schwartz has reported finding hemorrhagic lesions of the brain in 65 per cent of cases coming to autopsy, the author points out the fact that in many cases the pathologic changes were demonstrable only by histologic methods. One may properly speak of a physiologic birth trauma which does not leave clinical after effects. The true relation of late disturbances, such as idiocy, epilepsy, Little's disease and paralysis, to intracranial injury is at present undetermined.

The author believes that in the majority of instances gross examination of the brain will disclose any changes severe enough to be factors in causing death. Tentorial tears are the most frequent and fatal of the various types of lesions. They are usually associated with extensive subdural hemorrhage, and they may even occur in normal or cesarian delivery. Bleeding into the posterior

chamber is a characteristic lesion in premature infants. Leptomeningeal hemorrhage is not common. Concerning bleeding into the ventricles, Heidler has seen only five cases in six and a half years.

With regard to etiology, the author accepts in part only the explanation that the hemorrhage is due to a sudden release at birth of the excessive pressure to which the skull is subjected during labor. This, he thinks, may account for small or physiologic hemorrhages, but the more serious changes, tears, in particular, are due to mechanical factors. Among these he cites pressure of the bony pelvis and the uterine contractions on the head, operative maneuvers, etc.

The clinical picture may be indefinite. Asphyxia and irregular heart action are common. Absence of crying at birth suggests a tentorial tear. Moaning, apathy, cyanosis, convulsions, increased reflexes and a bulging fontanel occur in severe injuries, but even in these the autopsy may disclose nothing, unless one accepts Schwartz's histologic evidence.

To avoid injuries to the head at birth, Heidler recommends the avoidance, as far as possible, of all forcible methods of delivery and resuscitation of the infant.

OLMSTED, Seattle.

RESUSCITATION OF APPARENTLY DEAD NEW-BORN INFANTS BY THE ELECTRIC CURRENT. F. ISRAEL, Ztschr. f. Geburtsh. u. Gynäk. **91**:602 (Sept.) 1927.

Twenty per cent of asphyxiated new-born infants die. The author urges the use of the faradic current for resuscitation. An electrode, 10 mm. in diameter, is placed on each side of the neck over the phrenic nerve, and the other pole is placed over the fourth or fifth rib (level of the diaphragm) in the anterior axillary line of the corresponding side. The current is passed for two or three seconds at intervals of fifteen seconds. Such stimulation of the phrenic nerves primarily affects the diaphragm. Afferent fibers in the phrenic and vagus nerves carry stimuli to the respiratory center. Circulation and the action of the heart are improved by the negative pressure produced in the thorax. Electrical stimulation has many advantages over the more forceful mechanical methods. Reduction of the temperature of the body by cooling and rupture of the delicate blood vessels are avoided.

Six cases in which severe asphyxiation took place are reported. In these, medication with drugs was of no avail. Four stimulations with the faradic current at fifteen second intervals sufficed to induce regular respiration and heart action.

BRAHDY, New York.

Acute Infections

THE HEART AFTER SEVERE DIPHTHERIA. DUCKETT JONES and PAUL D. WHITE, Am. Heart J. **3**:190 (Dec.) 1927.

This study was undertaken to establish the relative importance of diphtheria as a factor in subsequent heart disease. One case of complete heart block that had persisted nineteen years after diphtheria at the age of 2 to 3, and another of complete heart block that developed twenty-three days after pharyngeal diphtheria, a few days after which auricular fibrillation appeared, are cited as suggestive. In this series, 100 patients were examined from five to eight years after a severe (70 per cent) or moderately severe (30 per cent) diphtheria. In twelve patients, the condition was diagnosed as myocarditis on the basis of split, weak, doubled or muffled sounds, gallop rhythm or extrasystoles. Of these, seven had apparently nonorganic systolic murmurs at the time of the diphtheria, and at the time of the reexamination four still had functional systolic murmurs. Evidence of organic heart disease was not found by history, physical examination, routine electrocardiogram or the roentgen ray (when needed). From this study there is no evidence of appreciable chronic effect of diphtheria on the heart.

MANNING, Santa Barbara.

THE PATHOLOGIC ANATOMY OF PERTUSSIS, WITH SPECIAL REFERENCE TO PNEUMONIA CAUSED BY THE PERTUSSIS BACILLUS. L. W. SMITH, Arch. Path. **4:** 732 (Nov.) 1927.

In a review of eight cases that came to autopsy in a series of about 3,000, the common complications found were lymphocytosis, peribronchial thickening, enlargement of the tracheobronchial lymph nodes and the occurrence of meningeal, conjunctival and other hemorrhages. The more unusual complications were the rare incidence of subcutaneous emphysema, intussusception, and otitis media caused by the pertussis bacillus.

In eight cases that came to autopsy with a complicating bronchopneumonia, many extensive changes were noted in the lung. These included bronchiectasis, fibrosis, and even a pseudometaplasia of the bronchial epithelium from the columnar to the squamous type. The importance of the pertussis organism as a primary etiologic factor in the mortality rate is emphasized.

SANFORD, Chicago.

AN ANTIMEASLES DIPLOCOCCUS SERUM. RUTH TUNNCLIFF and BENJAMIN WHITE, Boston M. & S. J. **197**:272 (Aug. 18) 1927.

This paper is a preliminary communication describing the production in the horse of a specific immune serum by the employment of the green-producing measles diplococcus of Tunnicliff, following the method described by Dochez for the production of antiscarlet fever serum.

This serum has been concentrated and when tested in both unconcentrated and concentrated states was found to contain opsonins and to produce neutralizing and protective effects specific for this diplococcus. Administered by injection to children previously exposed to measles, it apparently gave complete protection in a few cases, incomplete protection in some and no protection in others. It is possible that the dosage used was inadequate. Further clinical tests are being made with this concentrated serum.

INCIDENT OF CONTAGIOUS DISEASES IN SANATORIA. EARLE C. WILLOUGHBY, Boston M. & S. J. **197**:422 (Sept. 15) 1927.

The material for this paper comes from experiences at Massachusetts state sanatoriums which are caring for children. Conclusions are as follows:

Measles is extremely contagious and spreads rapidly when there is direct contact. Past history of the disease is unreliable. The best prevention is prompt isolation as soon as the child shows elevation of temperature, without waiting for an eruption.

Mumps loses its virulence as it spreads in an epidemic in a sanatorium. It is difficult, if not impossible, to prevent the disease from spreading from one unit to another. The disease spreads slowly in the same ward, and all susceptible children may not be infected.

Whooping cough and chickenpox do not seem to be troublesome diseases in sanatoriums, possibly because part of them have been fortunate in not having the disease brought in.

Scarlet fever does not have a great tendency to spread from one ward to another. It has a fair spread in the same wards in which the children are under 8 or 9 years of age and spreads less when the children are older.

One great difficulty in preventing the spreads is that some cases are mild and are not recognized until after the infective period is over.

Experiences with the Dick test and of giving antigen to a few children are recounted.

Diphtheria is not a problem in an institution in which Shick testing and immunization are carried out.

GENGENBACH, Denver.

BULLOUS IMPETIGO COMPLICATING VARICELLA. L. A. ELDRIDGE, JR., and T. M. RIVERS, Bull. Johns Hopkins Hosp. **41**:354, 1927.

Eldridge and Rivers report a case of chickenpox complicated by a bullous impetigo. *Staphylococcus aureus* was obtained from the bullous lesions and from a discharging ear. The condition was successfully treated with gentian violet.

LYTTLE, New York.

THE INTRADERMAL TEST FOR THE VIRULENCE OF DIPHTHERIA CULTURES. CHARLES S. GIEBS and CHEN KEN, J. Immunol. **14**:137, 1927.

The intracutaneous test for preliminary standardizing of diphtheria toxin and for determining the virulence of diphtheria cultures gave satisfactory results.

ARCH. PATH.

THE PRODUCTION OF DIPHTHERIA ANTITOXIN. ARTHUR LOCKE, E. R. MAIN and F. A. MILLER, J. Infect. Dis. **41**:32, 1927.

The preparation of diphtheria antitoxin serums of high titer requires that the quantities of toxin injected during the course of immunization be large enough to maintain maximum production. An interruption of maximum production may be detected at once by a rapid decrease from the normal in the flocculation time of a trial bleeding. The flocculation time of the plasma ordinarily passes through a minimum value of about six minutes during the first months of immunization and then rises gradually to a value of about two hours at the end of the third month, although it may be maintained at the minimum value by restricting the amounts of toxin injected to quantities which just constitute an antigenic stimulus, or it may reach values upward of four hours when the animal is being undesirably forced.

The avidity of a fresh antitoxin plasma, per unit of titer, is a function of the shortness of its time of flocculation with a given toxin. It has been evaluated for the plasmas drawn during this study and has varied from a maximum of 0.8 to a minimum of 0.5. Correspondingly, it has been found necessary to employ approximately 1.25 L_o units of the former preparation and 2.0 L_o units of the latter preparation to neutralize the toxicity of one L_o unit of toxin when the two were injected intravenously into rabbits immediately after mixture. When the times of injection of the toxin and antitoxin were separated by five minutes, approximately 1.25 x 200 L_o units of the first preparation and 2.0 x 200 L_o units of the latter were required.

AUTHOR'S SUMMARY.

MEASLES. JOHN H. MUSSER, M. Clin. N. Amer. **11**:619 (Nov.) 1927.

Observations made during a recent epidemic of measles have shown that new-born infants are not necessarily immune to the disease. Mothers who had had measles apparently conferred immunity on their children. On the other hand, in six instances, infants of mothers who were suffering from measles or who developed it shortly after parturition had the disease when they were born or developed it shortly afterward.

The complication of bronchopneumonia has to be diagnosed many times in the absence of any detectable areas of consolidation, even in cases that show extensive solidification at autopsy. It is recognized by the persistence of fever and cough past the normal period, and the presence of râles.

Encephalitis is apparently an extremely rare complication of measles, yet it was definitely proved to have occurred in six cases which came to autopsy, and it is believed to have been present in six others. The outstanding symptoms were associated with irritability and apathy. High fever occurred in all cases, the temperature often reaching 107. Involvement of the cranial nerves was not noted. Commonly there was more or less general spasticity of the peripheral muscles, and only when this was present did rigidity of the neck occur.

Examination of the spinal fluid showed: increased pressure, increased globulin and increased cell count, polymorphonuclears predominating. At autopsy, the brains were softened, with minute hemorrhages scattered throughout the gray and white matter, with occasionally a larger hemorrhage. The meninges were intensely injected but there was not an actual exudate.

SCARLET FEVER. CHARLES W. DUVAL, M. Clin. N. Amer. **11**:631 (Nov.) 1927.

From an acutely exudative focus, usually primary in the throat, the toxin of scarlet fever causes extensive parenchymatous degeneration in various important organs of the body. Perhaps the most important of these distant lesions is nephritis, which occurs in two forms, a primary or glomerular type, and a secondary type involving the interstitial tissues. In the causation of acute degenerative changes in the heart muscle, the acute nephritis of scarlet fever is more important than the specific toxin of the disease.

Although the streptococcal etiology of scarlet fever is now generally admitted, the exact nature of the toxin is still imperfectly understood. There is considerable experimental evidence to show that the toxemia is caused by the endotoxic product of the streptococcus, and that a soluble toxin is not extant at any time to account for the condition. Presumably two toxemic periods occur during the course of the disease. The early toxemia occurs in the first stage and is due to the action on the tissues of the whole protein of the specific streptococcus and, as a result, the host is stimulated to produce a bacteriolysin. This lysin in turn exerts a clevage action on the living streptococcal cell, and in consequence it is destroyed and an endotoxic product is liberated. Thus a second toxemic state is produced.

Experiments by the author show that a more potent serum for scarlet fever and other streptococcal infections should be produced with antigens that are essentially stimulators of neutralizing antibodies. Such antigens are the in vivo prepared streptococcal lysates which produce antitoxin in greater amount than is the case with living, killed culture or culture filtrate.

The employment of the in vivo prepared toxic product of the scarlatinal streptococcus offers a better means of obtaining a more potent antitoxic serum than is obtained with the use of either culture-filtrate (Dick's method) or killed or living culture (Dochez's method). The active streptococcal principle obtained by the method described by Duval and Hibbard and called by them "lysate" causes only the production of an antitoxic serum, while the whole streptococcus gives rise to the production of a lytic or bactericidal serum.

SMITH, Ogden, Utah.

ASSOCIATION OF MEASLES AND DIPHTHERIA. P. JOANNON, Progrès méd. **53**:1610 (Oct.) 1927.

The Schick test was made in 115 patients with measles and in 42 controls. Positive reactions to the Schick test were present in 40 per cent of the patients with measles and in 7 per cent of the controls.

Diphtheria that follows measles or accompanies it is more severe than measles appearing after diphtheria. In general, the organism that has been in contact with the measles virus contracts typical measles on exposure. A previous attack, however, protects against measles.

The receptive value of man to diphtheria is relative. There is a threshold varying from one person to another, and in the same person from one time to another.

SANFORD, Chicago.

COMPLICATION FOLLOWING MEASLES. A. CORBIA, Clin. Pediat. **9**:779 (Oct.) 1927.

Following an attack of measles, a child, aged 9 years, developed purpura hemorrhagica. The author believes that the complication occurred because the patient was constitutionally predisposed to it, due to acquired and con-

genital causes. The child had congenital heart disease with general vascular incompetence, a low platelet count, disturbed splenic function occasioned by a previous attack of malaria, a positive Pirquet test with adenopathy and an insufficiency of the suprarenals.

ZAMBARANO, Northville, Mich.

DIPHTHERITIC INTOXICATION AND MUSCULAR LESIONS. P. POLETTI, Riv. di clin. pediat. **25**:609, 1927.

The author has observed the histologic changes in muscle tissue in patients with diphtheria and postdiphtheritic paralysis. He believes that these changes (especially fatty degeneration) are not to be neglected in explaining the asthenia and paralysis in diphtheria.

HIGGINS, Boston.

THE FATE OF CHILDREN INFECTED WITH GONORRHEA. A. FRANK, Monatschr. f. Kinderh. **36**:483, 1927.

The name gonorrheal vulvovaginitis is a misnomer because in infants the rectum and urethra are also likely to be involved, but the vagina as a rule is affected most severely. Whether the infantile type has any influence on the future health of the woman is an unsettled question. The author reviews statistics quoted by a number of observers who have decidedly conflicting opinions. The difficulty of obtaining such statistics is obvious. The author reviews the cases of 259 patients treated in the Leipzig dispensary between 1900 and 1920. He concludes that the disease does not lead to complications in after life nearly as frequently as has been supposed. Of 110 women examined, salpingitis, endometritis and oophoritis were found in only five cases. In addition, it must be remembered that the treatment for the condition in infancy was not as satisfactory twenty years ago as it is now. Probably today the results would be even better.

ENLARGEMENT OF LYMPH NODES IN GRIP. GRETE WESTPHAL, Monatschr. f. Kinderh. **36**:506, 1927.

Westphal presents case reports describing a number of children suffering with grip complicated with unusual enlargement of the cervical lymph nodes. All recovered without suppuration.

INVESTIGATIONS CONCERNING THE NEGATIVE PHASE IN DIPHTHERIA INOCULATIONS. WERA KRESTINSKI, Monatschr. f. Kinderh. **36**:513, 1927.

Some investigators have maintained that there is a negative phase following inoculations with toxin-antitoxin. In a study on ten children, the author does not find the consideration of a negative phase of any practical importance.

GERSTLEY, Chicago.

INFANTILE INFLUENZA (GRIP). LEOPOLD MOLL, Wien. klin. Wchnschr. **40**:1486 (Nov. 24) 1927.

Moll believes that *B. influenzae,* although a serious causative agent in infantile grip, is rarely encountered in that disease. *Streptococcus hemolyticus, Micrococcus catarrhalis* and *"Pneumococcus planus"* of Adam head the large list of pathogenic organisms which he has found in these cases.

The author has observed grip in two forms. The simpler one is catarrhal and begins with nasopharyngitis and hypersecretion of mucus. The infant may assume a position of opisthotonos in order to breathe more freely. The inflammation often extends to involve the cervical lymph nodes, the cavities of the middle ear, the larynx (croup) and trachea. While occasionally primary in grip, bronchitis is usually secondary. When pneumonia develops, the classic physical manifestations are usually absent, because the process is paravertebral.

Anorexia, vomiting and loose, slimy stools indicate an intestinal form of grip, but Moll advises caution in making this diagnosis. In the treatment of catarrhal grip, he employs sweats, aspirin (from 0.1 to 0.2 Gm.) in strong tea, epinephrine or 1 per cent collargol instillations into the nose. One should try to prevent gastro-intestinal complications, but if they occur, an effort should be made to maintain the weight. The author uses quinine sulphate and urethane in influenzal pneumonia.

In the severe type of grip, an infant may be dead in two days without having shown catarrhal symptoms. Young infants are usually attacked. They display pallor, weakness, cold sweats, hypothermia or hyperthermia, attacks of dyspnea and cyanosis. Nervous symptoms are frequent. The symptoms in the lungs are minimal and variable. The liver is usually enlarged. According to the author, at autopsy one finds an intense congestion of the liver, portal vessels and lungs. This would suggest an "internal bleeding" resulting from vasomotor paralysis. Moll and Luksch found little if any epinephrine in the suprarenals of some of these patients. They are not prepared to say whether the deficiency of epinephrine or the toxin of grip is the cause of the vasomotor paralysis. Moll advises that the severe form of grip be treated by vasomotor stimulation. He employs hot mustard baths, epinephrine (up to 0.5 cc. of 1:1,000 solution subcutaneously) and caffein (0.5 Gm. by mouth, or from 0.5 to 1.0 cc. of 10 per cent solution subcutaneously).

OLMSTED, Seattle.

Chronic Infections

RADIUM THERAPY IN TUBERCULOUS CERVICAL ADENITIS. G. ALLEN ROBINSON, New York State J. Med. **27**:717 (July 1) 1927.

Twenty-five cases are reported: eighteen patients recovered, five were improved, one adult died of pulmonary tuberculosis and one of influenza. Of five children in this series, four are clinically well and one improved. Unless sinuses were present, scars did not remain.

TUBERCULOSIS OF THE ABDOMINAL LYMPH NODES IN EARLY LIFE. JOHN LOVETT MORSE, New York State J. Med. **27**:773 (July 15) 1927.

In the majority of cases recovery probably occurs without the condition ever having been suspected.

As there are no characteristic symptoms, the diagnosis is not easy. It may be made if the condition is kept in mind, especially in irritable children showing loss in weight, slight rise in temperature and pallor, especially if abdominal pain is present and the tuberculin reaction is positive. The enlarged glands can often be palpated. Roentgenograms are of little value.

Tuberculosis of the retroperitoneal glands is likely to be mistaken for appendicitis. In doubtful cases, it is well to operate.

In one case, an abscess the size of any eggplant developed from the mesenteric glands. Abscesses from glands may be mistaken for an appendical, perinephritic or psoas abscess.

The only patients in the author's practice that have died have been infants.

If Rollier treatment cannot be given, the mercury vapor or carbon arc lamps may be used. It is important not to trust too much to lamps and to forget that recovery is aided by air, rest, food and sunlight. Too much credit is given to ultraviolet rays from lamps. In the Rollier treatment, other rays of the sun and the stimulating action of the wind may be valuable factors.

AIKMAN, Rochester, N. Y.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS. G. ELEANOR SOLTAU, Brit. J. Tuberc. **21**:137 (July) 1927.

This method of treatment is much the same in children as in adults, and the same complications arise as in older patients. The type of case in which

artificial pneumothorax has been used has been almost entirely that which is called the "adult type," i. e., patients suffering with cough, positive sputum and some degree of fever. Children make good patients for this form of collapse therapy, after they have once gotten over the fear of the "pricks." Treatment in most cases does not have to be continued for long periods, and the pneumothorax is likely to come to an end spontaneously through collapse of the chest wall and the lung gradually becoming adherent. Even partial collapse seems highly beneficial in promoting arrest of the disease.

SMITH, Ogden, Utah.

THE DIAGNOSIS, TREATMENT AND PREVENTION OF TUBERCULOSIS IN CHILDHOOD. J. A. MYERS, Tubercle **8**:515 (Aug.) 1927.

Myers sketches the history, aims and methods of the Lymanhurst School for Tuberculous Children in Minneapolis.

It has been found that the children examined in the outpatient department may be grouped as follows: 1. Children free from symptoms without abnormal physical or roentgenographic manifestations and with negative cutaneous tuberculin reactions are said to be entirely negative to tuberculosis and tuberculous infections. 2. Children free from symptoms without abnormal physical or roentgenographic manifestations but with positive cutaneous tuberculin reactions are said to have tuberculous infection without evidence of tuberculous disease. 3. Children with slight or no abnormal physical or roentgenographic manifestations with positive or negative cutaneous reactions are classified as tuberculous suspects. 4. Children with definite symptoms and without abnormal physical or roentgenographic manifestations but with positive cutaneous reactions are said to have masked juvenile tuberculosis. 5. Children with definite symptoms, with enlargement of the lymph nodes and with positive cutaneous reactions are said to have tuberculosis of the lymph nodes of the involved region. 6. In children with definite symptoms and with positive physical and roentgenographic signs in various parts of the body, a diagnosis of tuberculosis of the part involved is made.

Great emphasis is laid on the condition of the child as a whole, and on the recognition and treatment of any physical or mental ailment that the child may have aside from tuberculosis. It is recognized that the tuberculous child is much more likely to resist successfully its specific infection if the handicap of other abnormal conditions is removed.

PROPHYLAXIS OF TUBERCULOSIS IN INFANCY. LANGER, Rev. internat. de l'enf. **4**:648, 1927.

The fight against tuberculosis in childhood has been intensified in Germany since the war through the better organization of the various institutes of hygiene. Two objectives are considered, the first embracing all those measures known to be helpful in the protection of children threatened with the disease and the second concerned with the management of those already affected. Langer devotes the major part of his discussion to the first and most important phase of the subject. The well known incidence of infection among children up to 14 years, amounting to as high as 80 or 90 per cent in large cities, is sufficient evidence to prove that it is neither possible nor, happily, even necessary to attempt absolute protection of the child population; the important thing is to take proper cognizance of the rôle played by the two chief factors in contamination — exposure and disposition. It has become a trite expression among epidemiologists that the earlier a child is exposed to tuberculosis, the greater the danger, as demonstrated by the high mortality of certain families in which it is doubtless due either to household infection or to insufficient equipment in combating disease. It is also recognized that the menace of repeated contacts at the period of puberty is particularly great. To prevent, so far as can possibly be done, these yearly recruits to the army of consumptives

in Germany, the central bureau of national associations for social hygiene in Berlin has issued a series of bulletins covering every phase of the problem. Not only the housing situation, but the importance of diet, the value of meals at school, the cultivation of physical strength and prowess and the necessity of fresh air and sunlight are all adequately stressed. The bureau recommends removal of patients to solariums or seashore institutions far removed from the holiday crowd. Vaccination either by the Calmette method of using attenuated cultures or by Langer's method of killed cultures is mentioned as the most hopeful type of treatment yet devised.

AMESSE, Denver.

SEDIMENTATION RATE OF ERYTHROCYTES IN INFANTILE TUBERCULOSIS. F. PARADISO, Clin. Pediat. **9**:723 (Oct.) 1927.

One hundred patients who had various types of tuberculosis were studied with reference to the sedimentation rate of the red blood cells. The cases varied from those merely showing positive skin reactions to those presenting active and widespread lesions. There was a diminution in the time of sedimentation in the graver forms of the disease when the test was analyzed in a general way. On more careful study of the individual cases, this parallelism between the sedimentation rate and the progress of the disease became less marked. In many infants whose general condition was excellent and who only had a positive skin test, the rate was more rapid than in patients with advanced disease and poor nutrition. This fact renders the sedimentation test practically useless as a means of determining the activity or prognosis of the disease.

ZAMBARANO, Northville, Mich.

THE SOCIAL IMPORTANCE OF SYPHILIS IN THE CHILD. H. DAVIDSOHN, Gesundheit. f. d. Kindesalter **1**:581 (Aug.) 1926.

Statistics from all over the world (Germany, Czechoslovakia and North America) show that there are about from 2 to 5 per cent of congenitally syphilitic infants. This agrees with Davidsohn's figures and also with the established morbidity among pregnant women, which is between 4 and 11 per cent. Naturally, the figure must be somewhat higher since a large percentage of persons die because of fetal syphilis. The disease occurs more frequently in the lower classes socially. The following statistics of Pfaundler are interesting: 60 per cent of the infants of syphilitic mothers died in the first year, and only 5 per cent remained free from all physical signs up to the fourteenth year. These figures are in general agreement with the results of many other authors. Davidsohn's own mortality is 75 per cent. He finds that the mortality in institutions is greater than in the home. Three fourths of all the deaths in the first year occur in the first quarter. This phenomenon is also found in syphilitic children who remain in the home. Nassau finds the causes for this high mortality in the increased danger due to infections; the lack of breast milk which would increase the resistance and then the well-known dangers of institutional nursing. Nassau calls attention to the late signs of congenital syphilis—the so-called degeneration stigmas of Fournier, which have also been found by Friedrich Mueller, Husler and Toni Schmidt-Krapelin. Plaut found that there was a distinct tendency to a retardation of the development of the feelings and the will, which tended to give rise to the development of criminals and degenerates. Huebner found that from the fifth to the eighteenth year, 17 per cent of congenitally syphilitic children developed syphilis of the central nervous system. After the eighteenth year there was only an additional 2 per cent. The best treatment in congenital syphilis is prophylaxis. Every child who is born of a syphilitic mother, whether or not he has signs or symptoms, should be treated. The author, however, believes that more attention should be paid to the general condition of the child and that one should be conservative in the dosage in the beginning of the specific treatment.

REICHLE, Cleveland.

TWO CASES OF ACUTE DEGENERATION OF BRAIN AFTER MEASLES. KARL MOSSE, Jahrb. f. Kinderh. **112**:272 (May) 1926.

Mosse reports a case of simple measles in a girl, aged 5 years. The temperature was 40.2 C. (104.3 F.) at the time of the first appearance of the exanthem. Five days later fever was not present, and the subjective condition was good. In the evening, however, the patient felt dull. The next day the temperature rose, and there were tetanic spasms in the arm and a condition of deep stupor. The spinal fluid was under high pressure, clear, and showed only a small increase of albumin and no decrease of sugar. Some reflexes were obliterated. Typical signs of a basilar meningitis were lacking. The whole disease picture resembled encephalitis. The second case was in a boy, aged 5 years, who in the second week of whooping cough, developed measles. The measles ran a normal course and the temperature fell on the seventh day. Psychotic disturbances appeared, to which were added disturbances of the innervation of the bladder and intestine. Involvement of the vegetative nervous system was evidenced by sweating and dermographism. After four days without fever, the temperature rose; some of the reflexes were increased, others obliterated. Stupor increased, and the patient died. These were not cases of encephalitis, since there was no inflammation of the brain, but a decomposition of its substance. Whether the toxins came from the measles organism or from some other for which the measles agent merely set the stage can be determined only when the organism of measles and its characteristics are known.

PACE, Chicago.

SKIN TESTS WITH SUSPENSIONS OF KILLED SPIROCHAETA PALLIDA. WALTER POCKELS, Monatschr. f. Kinderh. **36**:501, 1927.

In 143 tests, the author found that the intradermal injection of a suspension of killed spirochetes was a fairly reliable test. However, it should never be used without a controlling Wassermann reaction. It is interesting that nine of eighteen mongolian idiots gave positive reactions. The author believes that the test will not be of great value until the fluid is freed of much unnecessary protein.

THE BLOOD PICTURE OF LATENT TUBERCULOSIS IN CHILDREN AND ITS PROGNOSTIC SIGNIFICANCE. O. GRISGOROWA, Monatschr. f. Kinderh. **36**:519, 1927.

The author dwells at some length on the importance of lymphocytes in resisting tuberculosis. She also speaks of the Arneth-Schilling means of classifying leukocytes and then suggests an index of her own, namely, the relation of the mononuclear-leukocyte-macrophages to the other leukocytes. She believes that changes in this index are of valuable prognostic importance.

A CASE OF JUVENILE PSEUDOTABES SYPHILITICA. EUGEN KRAMER, Monatschr. f. Kinderh. **36**:538, 1927.

Kramer describes the occurrence of this disease in a boy, aged 6 years. While clinically the picture is that of tabes, pathologically the changes are those of a circumscribed meningitis. Under such circumstances the disease is more amenable to treatment, and under an antisyphilitic cure the boy made a good recovery.

CONGENITAL SYPHILIS AND TESTS FOR LIVER FUNCTION. A. SOLOMON and L. SCHOENTHAL, Monatschr. f. Kinderh. **37**:15, 1927.

In infants with congenital syphilis, the bilirubin picture in blood serum was examined before, during and after intensive treatment with neoarsphenamine. As it did not show any deviation from the normal, the author believes that intensive treatment with neoarsphenamine is indicated.

GERSTLEY, Chicago.

MYOARSPHENAMINE IN INFANTS. RUDDER, München. med. Wchnschr. **74**:2143, 1927.

The use of myoarsphenamine instead of arsphenamine and neoarsphenamine is preferred for the treatment of congenital syphilis in infants because of the ease of injection. The injections are made in the gluteal muscles, which produces little if any reaction in the tissues or general reactions.

Ten injections have been given in conjunction with bismuth and are believed to have fulfilled the demands of a successful antisyphilitic treatment in the new-born.

SCHORER, Kansas City, Mo.

PRESENT STATUS OF BCG. RUDOLPH KRAUS, Wien. klin. Wchnschr. **40**:1508 (Dec. 1) 1927.

Kraus reviews his own investigations as well as those of other European workers on Calmette's "BCG" or modified tubercle bacilli for vaccinating infants against tuberculosis. He states that there is general agreement that intraperitoneal and intravenous inoculation of BCG in animals produces a strictly localized form of tuberculosis in from three to four weeks. This completely retrogresses within a few months. Kraus interprets the fact that BCG is capable of producing characteristic tuberculous lesions as proof that it possesses a certain degree of virulence. He considers it a vaccine in the true sense of Pasteur. He has been unable to demonstrate any increase in virulence of BCG after it has remained in experimental animals for long periods. Subcutaneous injections have proved unsatisfactory in the experience of Kraus; local abscesses have been produced without subsequent development of immunity.

The author is of the opinion that BCG should not be introduced into medical and veterinary practice until authoritative experimental, clinical and pathologic studies have been completed. He sees a need for ultimate state control of the manufacture and distribution of the product.

OLMSTED, Seattle.

TUBERCULOSIS OF THE URETHRA. H. KUDLICH, Ztschr. f. Tuberk. **48**:115, 1927.

In a boy, aged 5 months, a caseous cavernous primary focus in the apex of the left lower lobe and multiple hematogenous metastases were found. Special attention was given to a caseous focus, the size of a bean, in the corpus-cavernosum of the urethra. Since the rest of the urogenital tract did not show any tuberculous foci, a descending infection could be excluded. The urethral focus is interpreted as a hematogenous metastasis, and the case is reported on account of the rarity of this localization.

MAX PINNER.

THE PRIMARY COMPLEX IN TUBERCULOSIS OF CHILDREN. JOHN NAESLUND, Upsala Läkaref. Förh. **32**:299, 1927.

Lungs with the so-called "primary complex" lesions of tuberculosis were obtained from twenty children and young persons during five years at the Pathological Institute at Upsala. The disease in the lungs as well as the related disease elsewhere in the bodies is described with great detail. The average age was 5 or 6 years, with a variation of from 2 months to 21 years. Three patients had a definite history of exposure; eight others, a possible infection through relatives, and nine did not have a history of exposure.

The pulmonary disease was not evident during life from physical examination, and roentgenograms in five did not give evidence of primary foci. Lumbar punctures lowered the pressure of the spinal fluid from 60 to 110 mm. It had the characteristics commonly encountered in tuberculous meningitis in the nineteen patients who died with this complication. In nineteen of the twenty, the first infections had occurred in the lungs, thirteen in the right, five in the left, and one had a primary focus in both lungs; thirteen foci were subpleural,

six in the parenchyma. The corresponding lymph glands were greatly enlarged and caseous. In the twentieth patient, the tuberculosis had its origin in the bowel in an ulcer 70 cm. above the ileocecal valve. In fifteen others the intestine was without change, but in three, the mesenteric glands were caseous. Miliary tubercles were found in the spleen and liver in ten. The usual structural alterations of tuberculous leptomeningitis were present in seventeen (complete postmortem examinations were not obtained in two).

Grossly the primary foci were irregular, circumscribed regions of caseous pneumonia, which ranged in size from that of a millet seed to that of a dove's egg. Tuberculous cavities were rare and small. Microscopically the caseous portions were surrounded by granulation tissue and between these there was a zone that was homogenous and stained with eosin. The lymph glands had similar changes. There was beginning cicatrization of the primary focus in the lungs of a child, aged 11½ years, and complete scarring in those of a man, aged 21. In three the intestines had microscopic tuberculous changes.

Naeslund concludes, as others have, that in the lungs the disease begins as a minute region of pneumonia. It is apparent, when the disease progresses to systemic infection and death, that the changes in the lungs are similar to those of acute pneumonic phthisis developing later in life in persons who have at the same time one or more healed "primary complexes."

LOUISA BACON.

DIFFERENTIAL DIAGNOSIS OF EARLY PULMONARY TUBERCULOSIS AND MITRAL DISEASE. M. H. CHIEN, National M. J. China **13**:184 (April) 1927.

The author was led to the consideration of this subject because of the unusually high percentage of mitral disease seen in the tuberculosis clinic of the Hunan-Yale Hospital and the marked similarity of symptoms in these two conditions. Among 153 patients seen in this clinic, thirteen, or 8.4 per cent, had mitral disease. Chien reports cases which show the similarity of symptoms of early mitral disease and incipient pulmonary tuberculosis. He cites the difficulties of diagnosis, and the character of hemoptysis and sputum in mitral disease with pulmonary infarct is emphasized. The roentgen ray, he believes, is most important in diagnosis.

LUCAS, San Francisco.

Internal Diseases

DISPLACEMENT OF THE HEART IN PNEUMONIA IN CHILDREN. J. P. CROZER GRIFFITH, Am. J. M. Sc. **174**:448 (Oct.) 1927.

Griffith reported the study of forty cases diagnosed as pneumonia. He selected such subjects as were in good health before developing pneumonia and in whom the disease was acute, unilateral and apparently of a croupous nature for the determination of the position of the heart by roentgen-ray examination made early in the course of the attack. In cases in which cardiac displacement was found, a second roentgenogram was obtained before the child left the hospital. Of the forty cases, sixteen exhibited displacement of the heart to the affected side, and Griffith thought that the cardiac displacement was temporary. If delayed resolution is present, the return of the heart is delayed as well. The author concludes that a cardiac displacement is dependent at least primarily on distention of the healthy lung. The reports of seven of the cases are given in detail. The character of the disease in the majority of instances was croupous pneumonia, without any discoverable evidence of atelectasis. Griffith's report shows the relative frequency with which displacement occurred and that the condition is probably not at all infrequent.

THE AMOUNT OF BLOOD FOR TRANSFUSION. HAROLD W. JONES, Am. J. M. Sc. **174**:466 (Oct.) 1927.

Jones found that a satisfactory method to determine the dose of blood for transfusion did not exist and that as a result of his experience in nearly

2,000 transfusions, a dose of 400 cc. in primary anemia and chronic secondary anemia was sufficient for an average adult weighing 150 pounds (68 Kg.) and 5 feet 8 inches (172.7 cm.) in height. Most patients with secondary anemia require only from two to four transfusions to produce satisfactory results. Jones termed this a standard dose. He found that the method of dose depending on body weight alone was not satisfactory. The method he offers for determination of the dosage depends not only on weight but on the height of the patient; those of short stature who are greatly overweight do not receive more blood than those of less weight but greater height. He states that a tall person who is much under weight can accommodate a greater volume than estimated from body weight alone. The following formula is used:

Standard dose	400
Standard surface area	1.8
Unknown dose	x
Surface area recipient	0.2

The figure 0.2 is that of an infant 20 inches (50.8 cm.) long and weighing 8 pounds (13,628.74 Gm.). From the formula, the calculating is as follows:

$$1.8\,x = 80$$
$$x = 44 \text{ cc.}$$

The chart shows standard height, weight and body surface from birth to 19 years from which the determinations are made and also shows that if the patient is greatly overweight or underweight the surface area may be calculated by using normal weight for height and age instead of his own weight.

DIABETIC ACIDOSIS AND LEUKOCYTOSIS. FRANK N. ALLAN, Am. J. M. Sc. **174**:506 (Oct.) 1927.

Allan states that diabetic acidosis sometimes causes symptoms which closely simulate an acute intra-abdominal lesion, and the confusion caused by the misleading nature of the symptoms is increased by the leukocytosis associated with the acidosis entirely independent of the infection. He reports five cases of diabetic acidosis in which leukocytosis was found. In one case in which diabetic acidosis not only had clinical manifestations but in the occurrence of leukocytosis which was associated with appendicitis, the leukocytes numbered more than 60,000. In other patients with acidosis, the author demonstrated the occurrence of leukocytosis in the absence of infection. In these cases, leukocytosis varied from 16,000 to 28,000, but the count returned to normal rapidly following the disappearance of the acidosis.

HENSKE, Omaha.

THE NEOPLASTIC NATURE OF LYMPHATIC LEUKEMIA AND ITS RELATION TO LYMPHOSARCOMA. W. A. EVANS and T. LEUCUTIA, Am. J. Roentgenol. **15**:497 (June) 1926.

Experimental studies favor the infective conception of lymphatic leukemia. Ewing classified the acute form as infectious and the chronic as essentially neoplastic, but the clinical course of the latter strongly suggests the continued presence of an irritant of toxic or bacterial nature as the existing cause of the process. In three patients with lymphosarcoma, lymphatic leukemia resulted as soon as the bone marrow became involved by foci of lymphosarcoma. These three patients are the only ones showing lymphosarcoma in a group of sixteen treated with deep roentgen-ray therapy in whom the disease terminated fatally. In all three radiation produced prolongation of life, with entire disappearance of the manifest lesions treated, but later, extension to the bone marrow occurred. Thus, if life is sufficiently prolonged, lymphosarcoma may be changed to lymphatic leukemia.

LYMPHOBLASTOMA (HODGKIN'S DISEASE) OF THE STERNUM. RICHARD DRESSER, Am. J. Roentgenol. **15**:525 (June) 1926.

Lymphoblastoma of the sternum is nearly always seen as a complication of a more or less generalized lymphadenomatous process, most probably metastatic. Four cases of sternal tumor were found in ninety-five cases of lymphoblastoma observed in the past three years. By administration of small daily treatments, from one eighth to one third of an erythema dose, until a total of from one half to two thirds of an erythema dose has been given to one area, it is possible to avoid roentgen-ray sickness in practically all cases. In this way, treatment may be given over a period of several years without producing the slightest visible effect on the skin. The osseous lesions respond readily to irradiation, and their presence seems not to affect the prognosis of the usually fatal disease in any way.

STULIK, Chicago.

EXTRAMEDULLARY HEMATOPOIESIS IN ANEMIAS. DORSEY BRANNAN, Bull. Johns Hopkins Hosp. **41**:104 (Aug.) 1927.

The author reviews all available reports of cases of von Jaksch's anemia with necropsy and reports one case of his own with autopsy together with the case of an anemic adult showing extramedullary blood formations.

He concludes:

1. "Extramedullary hematopoiesis is a fairly common finding in certain anemias of infancy and childhood, and large tumor-like growths of hematopoietic tissue may occur, particularly in the hiluses of the kidneys.

2. "Blood formation outside the bone marrow is occasionally observed in the severe anemias of adults, even in the presence of a hyperplastic bone marrow. Hematopoiesis may be observed in the broad ligaments and in organizing thrombi, as well as in the usual sites.

3. "Extramedullary blood production is to be regarded as a compensatory reaction.

4. "Blood production may be observed in the broad ligaments and breasts of infants, under apparently normal conditions.

5. "In extramedullary hematopoiesis the process appears to start in foci of type cells, either erythopoiesis or leukopoiesis, which substantiates previous observations regarding blood formation in the bone marrow."

LYTTLE, New York.

ACHOLURIC FAMILY JAUNDICE. R. H. TALLERMAN, Proc. Roy. Soc. Med. **20**:62 (Aug.) 1927.

The case reported occurred in a female infant, aged 9 weeks, who was taken to the hospital on account of constipation and jaundice. The jaundice was present at birth; it disappeared, but reappeared in six weeks and since then remained constant. The mother suffered from jaundice all her life until she underwent splenectomy in 1925. The child's maternal grandfather and her maternal greatgrandfather had suffered from jaundice and bilious attacks. The physical examination and the blood counts were given in the report.

CASE OF STUNTED GROWTH. D. W. WINNICOTT, Proc. Roy. Soc. **20**:66 (Aug.) 1927.

The patient was aged 5 years, and weighed 21 pounds (9.5 Kg.). He always looked anxious. He had suffered from asthma. There was some degree of clubbing of the fingers, and marked epicanthic folds were present. Symptoms or signs pointing to renal or intestinal infantilism were not seen, and the facies did not remind one of any well described group. The Wassermann reaction was negative. Examinations of the blood and urine were normal. The author reported the case in order to find out the cause of the stunted growth.

SUBACUTE INFECTIVE ENDOCARDITIS IN CONGENITAL MORBUS CORDIS. BERNARD SCHLESINGER, Proc. Roy. Soc. Med. **20**:68 (Aug.) 1927.

The case reported occurred in a boy, aged 14 years. He complained of suffering from shortness of breath and pains in the knees for one month, at which time he had had an attack of influenza. Until then he had felt well, although it was known that he had congenital heart disease. The physical signs, the blood count and cultures made from the blood were given in the report.

WILLIAMSON, New Orleans.

TWO CASES OF FAMILIAL SPLENOMEGALY. ROBERT HUTCHINSON, Proc. Roy. Soc. Med. **20**:321 (Feb.) 1927.

The two cases reported were in brothers, aged 11 and 5 years, respectively. The parents and three sisters were healthy. Both patients had suffered from attacks of jaundice. Each had an enlarged liver and an enlarged spleen. Blood counts were normal. The Wassermann reaction in one was doubtful; in the other, it was negative.

WILLIAMSON, New Orleans.

HEREDITY IN SIMPLE GOITER. W. RUSSELL BRAIN, Quart. J. Med. **20**:302 (April) 1927.

A series of six families is reported including among them twenty-six persons with simple goiter. In two of these families, cases occurred in four successive generations. A second series of five families is reported, containing twelve cases of simple goiter associated with deaf mutism.

Evidence is brought forward in support of the view that hereditary predisposition is a factor in the etiology of simple goiter in some cases. It is concluded that simple goiter may be the manifestation of more than one abnormal hereditary factor, two of which can be distinguished by their behavior in transmission.

SANFORD, Chicago.

TREATMENT OF AN ANEURYSM IN A SIX YEAR OLD BOY. GEORGE CHIENE, Edinburgh M. J., Tr. Medico-Chir. Soc. Edinburgh **34**:145, 1927.

A puncture wound in the popliteal space was followed in a few weeks by a popliteal aneurysm which completely filled the space. The surgical treatment consisted of application of a tourniquet, the incision of the aneurysmal sac which was emptied and the artery sutured. The final technic of using the tourniquet is described. Uninterrupted recovery resulted.

NEFF, Kansas City, Mo.

A CASE OF LIPOID-CELL SPLENOHEPATOMEGALIA IN AN INFANT AGED SEVENTEEN MONTHS. E. R. SCHIFF, Jahrb. f. Kinderh. **112**:1 (March) 1926.

A boy was given breast milk for the first four months and was then fed on cow's milk. At the age of 5 months, he received vegetables and lemon juice. At 3 months the parents noticed the enlarged abdomen, and the child had never been well since that time.

Physical examination revealed marked pallor, with brown pigmentation on the face and hands and a bluish mongolian spot over the lumbosacral region; malnutrition, palpable epitrochlears, geographic tongue, snoring respiration, markedly enlarged abdomen (51 cm. in circumference in the region of the umbilicus), enlarged liver and spleen, both of which were smooth and firm and extended to the superior iliac of the spine, were present. Examination of the blood showed 70 per cent hemoglobin (normal, 80 per cent); from 4 to 6 million red cells, and 10,800 white cells; the differential count showed: 49 per cent lymphocytes; 3 per cent embryonic; 14 per cent polymorphonuclears with unsegmented nuclei; 31 per cent polymorphonuclears with segmented nuclei; 2 per cent monocytes and 1 per cent eosinophils. Polychromatophilia, aniso-

cytosis or poikilocytosis were not present. Examination of the blood showed: 200,000 platelets; normal bleeding and clotting time; normal osmotic resistance of the cells to hypotonic salt solution; 6.7 per cent albumin in the serum; cholesterol content, 0.31 per cent. The size of the spleen did not decrease following the injection of 0.5 cc. of 1:1,000 epinephrine. The tuberculin test was negative. The urine showed a few leukocytes, erythrocytes and epithelial cells.

The pale brown discoloration of the skin and the enlargement of the liver and spleen is seen in only two conditions—Gaucher's splenomegaly and lipoid-cell splenomegaly (Pick). In Gaucher's disease, the liver never enlarges as rapidly as the spleen and begins to enlarge only after the spleen has reached a marked development.

Puncture of the spleen in this case showed five or six typical large cells in each field, which contained lipoid and neutral fat.

THE CAUSE OF CYLINDRURIA IN INFANTS. AUGUSTA FUCHTWAUGE and MARIA LEDERER, Jahrb. f. Kinder. **112**:7 (March) 1926.

Cylindruria has been shown to occur in acid urine and to be decreased in alkaline urine. In order to find other factors influencing the occurrence of cylindruria, the urines were examined for specific gravity, hydrogen ion concentration and surface tension.

Cylindruria occurred frequently in infants who had sudden or slow losses of weight—probably a disturbance of water retention—and also, of course, in intoxication and diabetic coma. Pressure over the regions of the kidneys for ten minutes in infants likewise brought on cylindruria with the appearance of albumin, erythrocytes, and epithelial cells. Injection of calcium chloride and sodium chloride had the same effect.

Stalagmometric examination of the urine showed that normally the infant's urine is poor in colloid, that is, it has the same surface tension as water. In infants with large amounts of urinary sediment, the surface tension was decreased, due to increased colloid. The cylinders are considered by the authors to be a cell-colloid which in the presence of acidosis becomes fluid, passes through the urinary tubules and in the process becomes cylindric in form.

EHRMANN, Chicago.

SEVERE HYPERTHYROIDISM AFTER FEEDING FRESH THYROID IN HYPOTHYROIDISM. FRITZ MULLER, Monatschr. f. Kinderh. **36**:254, 1927.

There is a tradition that thyroid extract in excess may produce dangerous symptoms, but authentic cases of poisoning by thyroid extract are rare. Indeed, a review of the literature suggests the opposite. A case has been reported in which a child by accident took fifty tablets of thyroidin without any noticeable injury. The author, therefore, reports the following:

A child, aged 5½, showed symptoms of myxedema. For six months she had received daily injections of thyroidin, which had been decreased to once a week for the last two months. For the last two weeks, she took daily four pieces of fresh sheep's thyroid. Following an attack of mild nasopharyngitis, she developed a severe nervousness and constant muscular activity described as not resembling a chorea, but more like a condition in which the skin was itching severely. The pulse rate went to over 200, with normal temperature. For three days, she stayed in a semicomatose condition, the muscular movements being scarcely affected by two doses of 1 Gm. of ethyl carbonate (urethane), ergotamine tartrate (0.3 Gm.), together with atropine (0.2 Gm.) hypodermically, caused a slowing of the pulse rate to 142, but this was only temporary. Later, with a stethoscope, the rate could be made out to be from 220 to 240. The child seemed moribund, but under hourly stimulation of

camphor, strychnine and insulin, made a gradual recovery. The author does not believe that this was a case of paroxysmal tachycardia, because of the long duration, but thinks it a case of real thyroid poisoning.

OMPHALIC PERITONITIS. H. NEWHOUSE, Monatschr. f. Kinderh. **36**:266, 1927.

Newhouse reports the case of an infant, aged 18 days ,with omphalic peritonitis cured by laporatomy.

PNEUMOTHORAX THERAPY IN CHILDHOOD. B. PETRICONI-BERCHTOLD, Monatschr. f. Kinderh. **36**:269, 1927.

Of eight children with definite pulmonary tuberculosis, who had been treated for twelve and a half months in a sanitarium and by pneumothorax, five showed great improvement and two seemed cured. In the five who showed improvement, however, one could not consider them cured by any means. One case ended fatally.

The author believes that pneumothorax is the best treatment for active tuberculosis in childhood.

FATAL HEMOPTOSIS IN CHILDHOOD. E. J. SCHMITZ, Monatschr. f. Kinderh. **36**: 301, 1927.

Schmitz reports two cases of fatal hemoptosis in young children. Both were manifestations of secondary tuberculosis, one more of the caseous and terminal miliary type, the other of the chronic cirrhotic type with formation of cavities.

CHOLECYSTOGRAPHY IN CHILDREN. G. WEBER, Monatschr. f. Kinderh. **36**:315, 1927.

Weber gives a description of the technic and roentgenograms of photography of the gallbladder in children.

INTERRENAL DYSTROPHY. GEZA PETENYI, Monatschr. f. Kinderh. **36**:323, 1927.

Under the title of interrenal dystrophy, the author gives a case report of a boy, aged 3 years, who died of a hypernephroma. The striking feature of this case was the combination of secondary sexual characteristics common both to puberty and to later life. The definite sexual development belonged to the period of puberty. Hypertrichosis, however, is characteristic of a later age. This combination of manifestations leads to the term "dystrophy," rather than to the use of other terms such as "pubertas praecox," which suggest simply a single symptom.

INTERRENAL DYSTROPHY. LUDWIG PUHR, Monatschr. f. Kinderh. **36**:328, 1927.

A complete pathologic examination was made in the case reported. It is interesting to note that the thymus of the patient, a boy, aged 3, was in a state of evolution usually found at puberty. The author quotes three other cases from the literature and agrees with Petenyi that the term "dystrophy" is to be preferred.

A CASE OF LYMPHOGRANULOMATOSIS. W. CATEL, Monatschr. f. Kinderh. **36**:337, 1927.

A case of generalized glandular swelling is described of the extreme youth of the infant, 2½ months. The youngest patient previously described was aged 5½ months. The first impression was that the condition was malignant, but the child made a splendid recovery under injections of sodium cacodylate (0.5 mg., increased rapidly to 2 mg. per day). Treatment was continued for six weeks. Clinical improvement was marked, and sedimentation of erythrocytes increased from fifty-three minutes to five hours.

THE TREATMENT OF BRONCHOPNEUMONIA IN INFANCY. CARL STAMM, Monatschr. f. Kinderh. **36**:345, 1927.

The author recommends the intramuscular injection of dextrose in doses of from 20 to 50 cc. of a 10 to 15 per cent solution in pneumonia. That this is not without danger is shown by one patient who developed severe necrosis with formation of pus and gas and, eventually erysipelas. Death occurred.

HEREDITARY HEMOLYTIC ICTERUS COMPLICATED WITH ACQUIRED ICTERUS. STEFFY FEILENDORK, Monatschr. f. Kinderh. **36**:377, 1927.

The author reports an unusual case showing a combination of hemolytic and cholangitic icterus.

SPLENOMEGALIC CIRRHOSIS OF THE LIVER OCCURRING IN A FAMILY. JOSEF SCANTO, Monatschr. f. Kinderh. **36**:392, 1927.

In a family of ten, one child developed cholelithiasis and three others a splenomegalic cirrhosis of the liver of unknown etiology. Associated with the latter was a disturbance of the endocrine system. The report is interesting in view of the definite familial tendency.

GERSTLEY, Chicago.

A CURED CASE OF THE ADDISON SYNDROME IN A BOY AGED 12 YEARS. J. L. A. PEUTZ, Nederl. Tijdschr. v. Geneesk. **71**:3295, 1927.

After some introductory remarks on the difficulties in the explanation of the different symptoms of Addison's disease, the author describes a case of this illness, diagnosed eight years before in a boy then 12 years of age, who recovered. The occurrence at this age as well as the total recovery is rare in Addison's disease. The typical symptoms of the disease were present. The boy was treated in the hospital by daily subcutaneous injections of 0.5 mg. epinephrine, and two times a day by a subcutaneous injection of solution of arsenious acid (von Ziemssen) by the administration of the tincture of strychnine and compound tincture of cinchona. With regard to the anorexia the diet had to be changed often. For a long time the patient also obtained fresh suprarenals from the abattoir. After a period of eight years the boy is now in a fairly good condition. The pigmentations have become pale, though they are still visible.

S. VAN CREVELD, AMSTERDAM.

Nervous Diseases

THE USE OF IODIZED OIL IN THE ROENTGEN-RAY DIAGNOSIS OF SPINAL LESIONS. ERNEST A. SCHMIDT, Am. J. Roentgenol. **15**:431 (May) 1926.

A recent modification of the original "iodized oil" is reported. It is lighter than spinal fluid, ascends in the spinal canal, and is recommended for myelography and venticulography. It is not as radiopaque as the original and this reduces its value. Iodized oil is of value in the diagnosis and exact localization of tumors of the spinal canal. The author was also able to demonstrate, by injection of iodized oil into the spinal theca, neurologic conditions such as multiple sclerosis, injuries of the spine and subdural tumor, but these observations are not conclusive. The dangers are medullary lesion through faulty technic, transitory aseptic meningitis, and action of a foreign body.

STULIK, Chicago.

CONGENITAL TABOPARESIS, TREATED WITH MALARIA. W. G. WYLLIE, Proc. Roy. Soc. Med. **20**:325 (Feb.) 1927.

A boy, aged 13, had been healthy and of normal intelligence, until the age of 10. He then began to suffer from severe headaches, which became a daily occurrence.

His disposition altered, and he became backward at school, quarrelsome, irritable and at times violent. He was first seen on April 8, 1926, at the Maida Vale Hospital. At that time the Wassermann reactions with the blood and cerebrospinal fluid were strongly positive. On August 25, he received 3 cc. of malarial blood intramuscularly. After twelve days had elapsed, he had pyrexial attacks which were stopped by a single dose of quinine sulphate. He did not show any ill effects from the treatment, and the headaches disappeared.

Next, he received six injections of neoarsphenamine consisting of from 0.05 to 0.45 Gm. At that time he did not have headaches, was not irritable, was well behaved, but was still mentally backward.

The author believed that the treatment had brought about some amelioration of his trouble.

WILLIAMSON, New Orleans.

UNUSUAL FORM OF HYDROCEPHALUS. NORMAN M. DOTT, Edinburgh M. J. Tr. Medico Chir. Soc. Edinburgh **34**:139, 1927.

A brief report is made by Hamilton for Dott, at a clinical meeting of the society, of an unusual form of hydrocephalus in a child, the dilatation being limited to the left lateral ventricle. The cause of the blocking of the foramen of Monro had been an old inflammation of the ventricular lining. At operation through the region just below the left parietal eminence the septum lucidum was removed and communication was opened between the two ventricles. A previous hemiparesis affecting the right side of the face and right arm then cleared up, and the circumference of the head was reduced 4 cm.

NEFF, Kansas City, Mo.

THE SKULL OF THE MONGOLIAN IMBECILE. DAVID M. GREIG, Edinburgh M. J. **34**:253 (May); 321 (June) 1927.

In the May and June numbers of this journal an interesting and unusual study of the measurements and abnormalities of the mongolian skull will be found. The author has not been able to find any previous reference to the macerated skull with the exception of a description of one case by Dr. John Fraser. Incidentally, Greig believes that the "Mongol" should not be classed with the idiots, as the mental ability is much higher. Three skulls are described with drawings and numerous photographs, the May number of the journal containing twenty-five in all. The different aspects of the skull, the interior of the cranium, the jaws, teeth and vertebral articulation are described in detail.

As is well known these persons seldom reach adult life, and none of the skulls studied by Greig was from an adult. He includes the measurements of Fraser's case which was, however, that of a person dying at 40 years of age. Interesting comparisons of the specimens are made by showing superimposed skull tracings. In the June number the writer discusses the marks of difference between the skull of a mongolian and that of the microcephalic imbecile, the former having a general smallness but a proportionately large cranial capacity, nearly twice that of the microcephalic imbecile.

The writer concludes that mongolism is a defect of growth in fetal life, rather than in embryonic development. He finds that the characteristics of the skull can be summarized as the ready bony ankylosis of the synchondroses at the base occurring at the time of maximum brain growth; shortness of the head and relative increase in the interzygomatic breadth; the spongy layer in the cranial bones is poorly developed; the teeth, especially the upper and anterior ones, are irregular; the palate is flat, broad, but infantile; the maxillae and facial bones have not grown completely; the lower jaw is modified in shape; and an infantile condition of the bones of the skull and face is shown by the many anatomic peculiarities.

EXPERIMENTAL STUDY OF MENINGEAL PERMEABILITY DURING INFLAMMATION. MME. NATHALIE ZAND, Rev. neurol. **1**:473, 1927.

This is the second of a series of articles dealing with meningeal permeability and the protective barrier between the blood current and the brain. The author believes that this protection is supplied by the histiocytes of the reticuloendothelial system that are found in the superficial layers of the pia mater and also covering the choroid plexus, because in the normal animal, after the injection of trypan blue, only these cells are found impregnated. Such colored cells are not found penetrating along the vascular sheaths. Only when the injections are continued to the stage of intoxication is the protoplasm of the choroid epithelial cells discolored.

It had already been demonstrated that mechanical injuries to the pia mater could provoke penetration of the dye into the depths of the nerve tissue and into the epithelium of the choroid plexus. In this article are detailed some experiments on the behavior of the blue particles on injection after an acute meningitis has been provoked by the aid of cultures of pneumococcus. When such a culture was injected suboccipitally and followed twenty-four hours later by the injection of trypan blue, the dye appeared in the choroid plexus epithelium. By means of dilute cultures, chronic meningitis was produced, and on repeated injections of the dye a turbid bluish cerebrospinal fluid was obtained which contained 2,000 leukocytes per cubic millimeter, mostly polymorphonuclears but including a few mononuclear elements which contained the dye stuff. At necropsy all the organs, including the bone marrow, stained intensely, and a shade of blue was seen in the nerve tissue. The choroid plexus and the pineal gland, in particular, were markedly stained. On microscopic examination the blue granules were found in the usual locations, and also in the choroidal epithelium and in the perivascular spaces of those vessels penetrating into the brain tissue.

Thus, in the case of meningitis, the dye was found in two unusual locations, the cerebrospinal fluid and the deeper perivascular spaces. The origin of these histiocytes in the cerebrospinal fluid is evidently meningeal, because cells containing bluish granules were not found in the blood examined at various intervals after intravenous injection of the dye. The large mononuclear elements that appear in the cerebrospinal fluid in cases of chronic meningitis are evidently these histiocytes coming from the pia. The dye must penetrate directly from the vascular system toward the choroidal epithelium through the vascular walls insufficiently protected by the injured pia mater. That the nerve tissue proper is not stained is probably due to the fact that the nerve cell does not possess the faculty of absorption, whereas the epithelial cell does. The ependyma does not take up the dye under any of the conditions noted in these experiments. As shown in the experiments, meningeal permeability is increased in an inflammatory state, a fact long known in clinical medicine.

The conclusions reached are: "(1) inflammation of the meninges renders them more permeable; (2) the epithelium of the choroid plexus stains much more quickly in cases of meningitis; (3) trypan blue penetrates slowly into the cerebrospinal fluid, appearing only after several weeks of inflammation; (4) the morphologic elements of the cerebrospinal fluid which contain the dye are to be considered as free histiocytes."

FREEMAN, Washington, D. C.

THE PATHOLOGY OF THE CEREBRAL CORTEX IN IDIOCY. CHIKAZO INABA, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**:70, 1927.

The brains of two idiots were examined and in both the cerebellum was found larger than normal; the Purkinje cells were markedly diminished in number and in Nissl preparations not a single normal cell could be found; most

of the cells were atrophied, and others showed ballooning in which the dendrons participated. The atrophic cells assumed grotesque shapes. The dentate nucleus was unusually well developed, almost hypertrophied, and showed many more convolutions than normal; it was also relatively richer in cells. There were no evidences of sclerosis. The nerve fiber bundles appeared unusually sharp, although here and there some paler areas were observed. The molecular layer was completely discolored.

The cerebral cortex was much smaller than normal, this being more marked in the frontal than in the occipital lobes. All cell layers seemed to be affected, the inner one being the only one that appeared even approximately normal; the fourth layer was most affected, the number of cells being less than half the normal. The contrast between the second and fourth layers was striking, the poverty of cells being greatest in the second and least in the fourth. The cells themselves were definitely atrophic, small in size with loss of prolongations, and were changed in structure. If it had not been for the preservation of the nuclei, most of these pathologic cells would hardly be identified as functioning elements.

The nerve fibers were more involved than the cells. No tangential fibers were seen. Fiber radiation was indistinct in the sensory regions (acoustic, sensory and optic), and no heterotopies were seen. The pathologic process in the fibers extended from the peripheral to the central part of the brain. Although the diminution of the cellular and fibrous elements of the cortex was replaced by a relatively marked increase in glia, nevertheless the picture was not that of sclerosis. The diminution of the cellular elements gave rise to a diminution in the size of the convolutions which made the sulci appear wider and deeper.

Inaba believes that in idiocy the malformation or developmental disturbance does not affect the cerebrum and cerebellum alike, but that the cerebrum is affected primarily and the changes in the cerebellum are secondary. He believes that the brain has completely developed but has become apparently secondarily reduced. He supports his views by the appearance of the pia and of the cerebral vessels. The former shows nodular thickenings, which are not due to secondary dilatations but rather to primary antecedent inflammatory processes, the residua of which could be demonstrated in some areas. The vascular changes are not those noted in presenile conditions but resemble those observed in cerebral syphilis. This may account for the fact that many authors consider that syphilis is the etiologic factor in idiocy. Unfortunately, no good anamnesis was obtained in the cases reported, so that this question could not be definitely determined.

In the concluding part of the study is a description of a normal but relatively small brain from a small person of normal intelligence. The smallness of the brain was well compensated for by an increase in cells. The number of cells was actually higher than that given by Economo and Koskinas.

As a result of his study, Inaba concludes that the outer layers of the cerebral cortex are more essential than the inner for intelligence; also that it is chiefly the cells of the second and the external parts of the third layer, and the external Baillarger and tangential fibers that mediate the higher psychic functions. In general, this view corresponds with that expressed in the literature.

KESCHNER, New York.

A SIMPLE METHOD FOR DETERMINING THE SUGAR IN THE CEREBROSPINAL FLUID. SIEGFRIED SAENGER, Monatschr. f. Kinderh. **36**:281, 1927.

Saenger describes a simplified technic, depending on the reduction of copper sulphate and centrifugalizing.

GERSTLEY, Chicago.

A Case of Colimeningitis. Ilse Reinhardt, Monatschr. f. Kinderh. **36**:341, 1927.

Reinhardt reports a case of meningitis due to *B. coli* in an infant, aged 2 months. Possibly, injury to the brain by the forceps at delivery might have made a place of diminished resistance.

Gerstley, Chicago.

Complications in Puncture of the Sinus. L. Fabian, Monatschr. f. Kinderh. **36**:390, 1927.

The author had three fatalities in puncturing the superior longitudinal sinus for blood. In the first, autopsy showed the sinus to be 5 mm. to the right of the midline. The needle had missed this entirely and had entered an aberrant vein, causing death from cerebral hemorrhage. In two other cases, death followed thrombosis which originated at the site of the puncture.

Gerstley, Chicago.

The Sugar Content of the Cerebrospinal Fluid. Elsa Lagergren, Acta Paediat. **6**:414, 1927.

The author's determinations are summarized in the following table:

Condition	Lowest Value, Mg.%	Highest Value, Mg. %	Average Value, Mg. %	Number of Fluids Examined
Normal	0.038	0.090	0.061	34
Convulsions without meningeal inflammation	0.036	0.090	0.060	25
Epidemic encephalitis	0.041	0.092	0.059	25
Mumps, meningitis	0.040	0.075	0.059	8
Acute poliomyelitis	0.030	0.065	0.049	13
Serous meningitis	0.030	0.060	0.049	10
Tuberculous meningitis	0.001	0.100	0.036	63
Meningococcus meningitis	0.000	0.060	0.026	23

The increased sugar content which has been described in epidemic encephalitis is not confirmed by these observations. The author feels that the sugar content is of some diagnostic value in lymphocytic spinal fluids. A low sugar content favors the diagnosis of tuberculous meningitis, while a high sugar content renders this diagnosis improbable.

Holt, Baltimore.

The Study of the Spasmophilic Diathesis. IV. A Case of Spasmophilia with Hypoplasia of the Thymus and Parathyroids, E. Böttiger and W. Wernstedt, Acta Paediat. **6**:372, 1927.

A child, aged 4 months, who had been almost exclusively breast-fed, developed typical tetany and died during a prolonged spasm of the glottis. Only rudimentary bits of thymus and parathyroid tissue could be found at autopsy. The authors attach more importance to the deficiency of parathyroid than to that of the thymus.

Epilepsy with Myoclonus (Unverricht). J. Pelnar, Čas. lék. česk. **64**:1681 (Nov.) 1925.

A boy, aged 15, who has had attacks of petit mal since the age of 7, developed a nocturnal generalized convulsive tremor at intervals of from two to four hours without eneuresis at the age of about 10 years. Since the age of 13 he has had irregular unequal spasmodic convulsions of the body and extremities. Treatment included bromides and tonsillectomy with only temporary remissions. Because of the myoclonus of individual muscle groups without motor effect, clonic convulsions with motor effect and a lack of stereotyped motions, this condition is differentiated from chorea and tic. The electrical irritability was normal. The pathologic condition possibly involves a mechanism similar to the corpora amylacea found in chorea.

Stulik, Chicago.

ATTEMPTS AT PRODUCING EPILEPTIC AND HYSTERICAL ATTACKS BY DEEP BREATHING (HYPERVENTILATION). OTAKAR JANOTA, Čas. lék. česk. **64**:1717 (Nov.) 1925.

Producing epileptic seizures with camphor, alcohol, cocaine, strychnine or compression of the carotids may be dangerous to the patient, as well as unreliable. Foerster reported a series of forty-five epileptic patients in twenty-five of whom he was able to produce epileptic seizures after ten minutes of deep breathing in the sitting position. In only one of these, however, was the reaction obtained at each and every attempt. In the author's attempt to verify these observations the results were as follows: ten of fifty-one epileptic patients studied were unable to breath according to instruction, eleven habitually had a shallow respiration and thirty breathed normally. In the thirty an attack was not produced even after a period of from forty-five to sixty minutes of deep breathing. Typical attacks were produced in only two instances and both in patients who were shallow breathers. A reaction was not obtained on subsequent trials, but both patients developed an habitual deeper respiration. In five cases of hysteria studied one patient developed a hysterical seizure after from five to six minutes. Hyperpnea did not have an apparent effect on one alcoholic person. The increased electrical irritability was somewhat greater in the epileptic group, but the results are not marked enough for a definite statement to be made.

STULIK, Chicago.

Diseases of the Ear, Nose and Throat

BRONCHOSCOPIC TREATMENT OF LUNG ABSCESS. GABRIEL TUCKER, Am. J. Roentgenol. **15**:419 (May) 1926.

The first bronchoscopy in any case is a diagnostic one, and will give information as to the location in the lobe of the lung, extent of involvement, the amount and character of the pus, the condition of the bronchi in the affected area, and uncontaminated cultures can be taken for identification of the bacteria. Differential diagnosis between tuberculous and nontuberculous suppuration may be aided. Neoplasm can be determined by removal of tissue. The presence of dilatations, strictured bronchi, deformity of the bronchial lumen, deviation and fixation of the bronchi and abnormalities of bronchial movements can be definitely determined. Radiopaque material such as bismuth or iodized oil, 40 per cent, may be introduced into localized areas, and by pneumonography the presence of cavities, bronchial, dilatations and strictures, and the relation of foreign bodies or pathologic processes in the lung to the bronchi can be definitely recorded on the film. In children, bronchoscopic drainage is carried out without anesthesia, general or local, and without sedatives. Local medication, such as mercurochrome-220 soluble, silver nitrate, gomenol and monochlorphenol can be applied directly. The author feels that the best result is obtained through the cooperation of the internist, roentgenologist, bronchoscopist and the surgeon.

GERSTLEY, Chicago.

THE TONSIL PROBLEM. E. R. ROBERTS, Arch. Otolaryng. **6**:565 (Dec.) 1927.

The tonsil is not a special organ with a special function, nor is the faucial tonsil a part of the digestive system. Because of the broken surface of its protective epithelium, its exposed position and the natural tendencies of its structure to attract micro-organisms, the faucial tonsil offers less resistance to the entrance of infection or the products thereof to any part reached by the circulating body fluids than any other point in the anatomy.

There are no rational contraindications to the surgical removal of the faucial tonsil per se, and evidence that a tonsil thoroughly removed ever grows is lacking.

SANFORD, Chicago.

THE "TONSIL" QUESTION. FRED STOKER, Lancet **213**:1125 (Nov. 26) 1927.

The author cautions against removing the tonsils if simple hyperplasia is present. If there are definite signs of obstruction in the nasopharynx only the adenoids alone should be removed, so also if they are responsible for a suppuration of the ear, chronic rhinitis, sinusitis, etc.

Enlarged cervical glands not demonstrably due to another cause are clear indications for operation on the faucial tonsils. Other indications are recurrent sore throats, and inflammatory conditions of the ears, pharynx and larynx.

The author makes three recommendations: 1. Neoplasms and blood dyscrasias should be excluded before what appear to be "just ordinary tonsils" are removed. 2. If the patient has a tendency to bruise easily, a blood coagulation test should be made. 3. The possible presence of congenital syphilis should be kept in mind when one is asked to remove adenoids for "catarrh" or nasal deformity.

LANGMANN, New York.

NASOPHARYNGITIS. H. HOCHSCHILD, Monatschr. f. Kinderh. **36**:219, 1927.

The author discusses the importance of nasopharyngitis in children. In a way he thinks that the increasing frequence is due to the changing climate. Formerly, seasons were well marked with periods of good weather between. At present, both winter and summer are becoming milder and the whole year's weather is becoming cloudy and humid. The hygienic condition of clothing and dwellings has shown little change in meeting these new conditions. The ventilation of the school and home is as miserable as ever. Overheated dwellings and schoolrooms are incubators of disease, and the clothing of children has not been modified for the new environment.

The author observes that the children with nasopharyngitis are inclined to have vasomotor disturbances and night sweating. One should be particularly careful to see that their clothing is kept dry.

The importance of the tonsils and adenoids is often overestimated. They are as frequently a result as a cause.

GERSTLEY, Chicago.

Diseases of the Skin

THE ROENTGEN-RAY TREATMENT OF KELOID. G. W. GRIER, Am. J. Roentgenol. **16**:22 (July) 1926.

Radiation is universally regarded as the treatment for keloid, although some prefer the roentgen ray, while others use radium. Uniformly satisfactory results are obtained in the treatment of keloids with unfiltered roentgen rays. The amount of radiation should not exceed 90 per cent of the erythema dose at any one treatment. In the estimation of the dose the area to be treated must be considered, because with small ports of entry the scattered component increases rapidly with an increase in field size. From six to eight weeks should elapse between treatments.

STULIK, Chicago.

ECZEMA IN INFANTS AND YOUNG CHILDREN. CHARLES GILMORE KERLEY, Canad. M. A. J. **17**:1173, 1927.

Kerley divides his patients with eczema into two groups: (1) the larger group in which the condition of the skin is due to protein sensitization and (2) the group in which the condition is due to local skin irritants.

The author's conclusions are that: 1. A given protein or proteins may cause eczema, and yet the skin may fail to respond to tests with protein. 2. The responsible food protein may often be rendered innocuous by subjecting it to prolonged heat. 3. Nonreacting animal food proteins are responsible for many of the eczemas of older children. 4. A patient with true infantile eczema who recovers under treatment of local applications alone has become immunized to the responsible protein factor.

MOORE, Omaha.

THE TREATMENT OF RINGWORM BY THALLIUM ACETATE. J. H. TIVISTON DAVIES, Proc. Roy. Soc. Med. **20**:70 (Oct.) 1927.

The secret of success with thallium lies in the after-treatment. In every case the infected hairs have to be extracted by hand on the day that the hair first becomes sufficiently loose. The author's practice was to give 8.75 mg. of thallium acetate per kilogram and to instruct the mother to rub Whitfield's ointment into the unaffected parts of the scalp and to wash the head every other day for the first week and every day for the second. As epilation progresses, the bald areas are painted three times a day with 4 per cent iodine in rectified spirit for four days and the whole scalp rubbed with sulphur ointment for the rest of the week, this sequence being repeated for about four weeks.

Only the slightest toxic effects, which consisted of pains in the joints of the lower extremities, occurred in normal children suffering with tinea after the application of thallium. Patients suffering from alopecia areata are hypersensitive to thallium.

WILLIAMSON, New Orleans.

ERYTHEMA NODOSUM AND RHEUMATIC CONDITIONS. ALBIN LANDAU, Acta Paediat. **6**:402, 1927.

Statistics are presented from the Gothenburg Children's Hospital on the incidence of erythema nodosum and various rheumatic conditions. During a period of four years, 130 patients with erythema nodosum and 136 with rheumatic disease (endocarditis, polyathritis or chorea) were admitted. In only five instances was erythema nodosum found in a child who also gave evidences of rheumatism. In three of these five children, the acute rheumatic manifestations were separated from the onset of the erythema by a period of several years. All of these five children had evidence of tuberculosis.

The age incidence and the seasonal incidence of the two conditions were also studied. Rheumatic manifestations were noted only twice in a child under 5 years of age, but thirty-five children, under 5 years, with erythema nodosum were observed. The difference in the seasonal incidence is not striking. Both conditions are more common in the winter and spring.

The author concludes from these statistics that the association of rheumatic disease with erythema nodosum is purely accidental.

HOLT, Baltimore.

THE PATHOLOGY OF STROPHULUS (LICHEN URTICATUS). J. AMBUS, Orvosi hetil. **71**:816, 1927.

Strophulus seems to be a skin eruption due to hypersensitivity. Two factors play an important part in its etiology, food and constitution. It may be that there is hypersensitivity to different kinds of food. The hypersensitivity to one kind of food may cease spontaneously, while to another it may continue. Febrile diseases may cause definite disappearance of strophulus. In some instances, change of diet does not affect the strophulus. An attempt was made to desensitize the child through parenteral injections of protein without any change of the diet, with apparent good results. In the vesicular type of strophulus, a great number of eosinophil cells were found. Half of these cells seem to come from the blood, while the rest seem to be histogenous in origin. At the height of the eruption or shortly afterward, changes were not found in the blood picture. Other forms of hypersensitivity manifested as urticaria present eosinophilia in the blood and in the tissues, while scarlet fever shows eosinophilia only in the blood.

TREATMENT OF FURUNCULOSIS IN INFANTS. Z. BOKAY, Orvosi hetil. **71**:1048, 1927.

Commercial streptococcus vaccine was used in one series of the cases and autovaccine in another series. The author concluded that vaccine treatment is nearly always effective if it is not forced too much, as infants will not stand

any strong reactions. The injections should be given every two or three days with increasing doses. The doses should be regulated so as not to give too severe local and general reactions.

ZENTAY, St. Louis.

Surgery and Orthopedics

THE END RESULT IN ALBEE'S OPERATION FOR SPLINTING VERTEBRAE. T. WINGATE TODD, Ann. Surg. **86**:837 (Dec.) 1927.

Todd reports the finding of an old splinting operation in a cadaver, but the clinical history was not available. A perfect functional result was obtained by means of a tibial graft covering the twelfth thoracic and first lumbar spinous processes. Where the splint was grafted into the spinous processes, there was marginal erosion and proliferation of new bone as in repair of a fracture. Where the splint had come in contact with soft tissues, proliferation had not taken place in the transplanted bone. It is concluded that a bone splint remains unchanged in its new surroundings and acts as a scaffolding for the erection of new bone tissue on itself.

JACKSON, Chicago.

SURGICAL CORRECTION OF CLEFT LIP AND CLEFT PALATE. GEORGE B. JOBSON, Arch. Otolaryng. **6**:434 (Nov.) 1927.

The author prefers to repair the lip first, this being accomplished as soon after the third week as possible, and not later than the fourth month if it can be avoided, for the following reasons: 1. The premaxilla and vomer bones begin to ossify about the fourth month, with the result that molding of them becomes progressively difficult. 2. Early closure of the lip will cause the margins of the alveolar cleft and palate to approach each other, and the premaxilla will be gradually forced into place in most cases by the constant muscular action of the lip. 3. Improvement in nasal breathing and in the facial appearance results. 4. There is less shock in consequence of the immature development of the nervous system. 5. Mental anguish of the parents is relieved by early repair of the lip.

Preliminary to the operation, fluoroscopic examination should be made of the thymus gland, and a blood coagulation test is necessary to guard against hemophilia. It is desirable to have the nutrition brought to the highest state possible.

At from 6 to 9 months of age, the first step palate operation should be performed, and one year later the final operation to make the parts complete before wrong habits of speech have become established.

SANFORD, Chicago.

TREATMENT OF EMPYEMA IN CHILDREN BY THE CLOSED METHOD AND SUCTION DRAINAGE. DAVID W. PARKER, Boston M & S. J. **197**:653 (Oct. 20) 1927.

Parker reports the results of operation on eighteen children under 10 years. Of these, two patients were under 1 year, six between 1 and 2 years, four between 2 and 3 years, two between 4 and 5 years, and four between 5 and 10 years. The only death occurred in a case of infection by *Streptococcus hemolyticus* in a child under 2 years. Death occurred in three hours after thoracotomy with a terminal temperature of 108 degrees. Procaine infiltration anesthesia was used in all except one case. Preoperative exploratory aspiration was always done. Operation was deferred if the fluid was not purulent. Details of operation and after-treatment with chlorazene or surgical solution of chlorinated soda (Dakin's solution) are given.

GENGENBACH, Denver.

CASE OF LATERAL SPINA BIFIDA. M. FITZMAURICE-KELLY, Proc. Roy. Soc. Med. **20**:73 (Oct.) 1927.

A case of lateral spina bifida was described in a girl, aged 4 years. She was first seen at the age of 1 month when a swelling of the left buttock and shortening of the left lower limb were noted. Later, roentgenograms showed a deficiency of the lateral mass of the sacrum and the fifth lumbar vertebra. She was lost sight of for some time. About a year previously, she returned with severe paralytic talipes of both feet and incontinence of urine. An osteotomy was performed, and it was thought that should she reach the age of 10, a stabilizing operation on the feet ought to make her fairly comfortable.

WILLIAMSON, New Orleans.

PYLORIC STENOSIS OF INFANCY. NELLY SEIFERT, Monatschr. f. Kinderh. **37**:45, 1927.

The author has evidently just discovered that there is a Fredet-Weber-Rammstedt operation for pyloric stenosis. She claims good results in twenty-nine cases.

GERSTLEY, Chicago.

TREATMENT OF SPASTIC PYLORIC STENOSIS IN INFANTS. C. FORAMITTI, Wien. klin. Wchnschr. **40**:1380 (Nov. 3) 1927.

Foramitti has observed a symptom complex in young infants which is identical with that of hypertrophic pyloric stenosis. Even the palpable pyloric tumor is present in some cases. At operation, however, in place of a hypertrophied pylorus, one encounters a compact and contracted organ which on histologic examination shows an increase in connective tissue rather than in muscle fibers. The author admits that this relaxed state may represent merely a transient condition at operation, and that the difference between the two types of pyloric stenosis is not a clearly defined one.

The author states that the Weber-Ramstedt operation has proved the most popular of the various surgical measures designed for the relief of pyloric stenosis in infancy. The chief objections to it are the occasional occurrence of postoperative hemorrhage and peritonitis, especially when the pyloric incision has not been covered by omentum or reflected pyloric muscle.

In an infant, aged 10 weeks, with spastic stenosis, Foramitti was unable to stretch the pyloric ring by digital invagination of the stomach wall. He therefore made a small incision on the anterior surface of the stomach and introduced a no. 14 size Hegar dilator into the pyloric canal. Recovery was rapid and uneventful. Subsequently, he performed this operation successfully in thirteen cases. Superficial tears of the pyloric serosa comprised the only complication. They occurred in seven cases and were easily repaired. The author believes that this internal dilation, which was first proposed by Loreta, is superior to the Weber-Ramstedt method on account of the freedom from postoperative hemorrhage and peritonitis.

OLMSTED, Seattle.

ABDOMINAL SURGICAL CONDITIONS IN CHILDREN. G. SALVIOLI, Clin. Pediat. **9**: 131, 1927.

This report is based on observations made on 7,000 children up to 9 years of age. Tuberculous peritonitis is the most chronic abdominal lesion in childhood. The diagnosis is rarely difficult except in acute forms. Among the acute types of peritonitis, that due to the pneumococcus was most commonly observed. In two cases, the abdominal lesion was secondary to a pneumococcic infection elsewhere in the body. Appendicitis was not common. It occurred in 3.41 per cent of the cases in which operation had been performed over a period of seven years. The course of the disease was usually favorable. A typhoidal etiology was established in one case. Acute invagination was seen in thirteen cases. Constitutional factors play an important part in its occurrence.

In two patients with intestinal obstruction, the lesion was due to helminthiasis, and in one, it was caused by a calculus mass composed of grape seeds.

Enterogenous infection did not play a part in the causation of tuberculous peritonitis in the patients observed. The ascitic form predominated. Appendicitis was confused with intrathoracic lesions, spondylitis and coxitis. Obstruction was diagnosed in patients with enterocolitis, tabes mesenterica and bronchopneumonia. Tuberculous peritonitis was diagnosed in patients with neoplasm and pyloric stenosis.

ZAMBARANO, Northville, Mich.

Miscellaneous

REPORT OF A CASE OF FATAL METHYL SALICYLATE POISONING. J. B. PINCUS and H. E. HANDLEY, Bull. Johns Hopkins Hosp. **41**:163 (Sept.) 1927.

This condition is rare, the authors finding only six cases recorded.

Ten hours after the drug (oil of wintergreen) was swallowed, there were frequent convulsions, a gradual development of cyanosis, Biot's respiration and death four hours later.

The blood showed an acidosis with a retention of phosphates. The nonprotein nitrogen was 62 mg. per hundred cubic centimeters and the protein was 6.5 per cent; calcium was 11.2 mg. per hundred cubic centimeters.

Autopsy was not performed.

LYTTLE, New York.

OUR OBLIGATION TO THE CHILDREN OF OUR COMMUNITIES. A. A. SHAWKEY, W. Virginia M. J. **23**:244 (May) 1927.

A plea is made for idealism in medicine and the wisdom is urged of giving one's undivided talents to attaining proficiency, and letting the financial considerations take care of themselves.

That one's obligation demands his very best, regardless of time, effort or remuneration, when dealing with the health and lives of individuals is stressed. Some instructions to parents at each visit is urged as a duty.

Superficial examinations, snapshot diagnoses, and haphazard treatments at small fees are condemned.

Abuse of free clinics and hospital lists is considered unethical and even dishonorable.

There should be absolutely no free medical or surgical treatment, but all such treatment should be paid for at regular professional rates from money provided by general taxes, to which the physicians would contribute their proportionate share. In this way the burden would be divided equally among all the people and not carried by a comparatively few physicians and surgeons.

AUTHOR'S ABSTRACT.

HEALTH EDUCATION IN PUBLIC SCHOOLS. A. FERNOS ISERN, Porto Rico Rev. Pub. Health & Trop. Med. **3**:95 (Sept.) 1927.

Whereas in early school life health habits are taught with the emphasis on their importance and methods, in high school the reasons for the health habits are explained. Causes of some of the more common diseases are discussed along with the best methods of control. Personal and civic, local and national health problems are dealt with according to their relative importance.

BARNETT, Chicago.

OUR BUREAU OF SOCIAL WELFARE. J. RODRIGUEZ PASTOR, Porto Rico Rev. Pub. Health & Trop. Med. **3**:108 (Sept.) 1927.

Under the department of health, a "Bureau of Social Medicine" has been formed by the consolidation of the services of maternity and child welfare, tuberculosis, venereal diseases, and the divisions of social welfare and public health nursing. This has resulted in greater economy and efficiency.

There are thirty dispensaries, about which the work of the organization centers. Traveling clinics for the rural communities are to be tried this year. The work is mostly educational, teaching the people general hygiene and prevention of disease. Practically all school children are given the Shick test, and toxin antitoxin is given when indicated. Infant welfare, visiting nurses, and social workers are all important factors in the organization.

BARNETT, Chicago.

ROENTGEN-RAY TREATMENT OF GLANDS AND BONES IN CHILDREN. L. FREUND, Wien. Klin. Wchnschr. **40**:1361 (Oct. 27) 1927.

Freund obtains good results within two or three weeks following repeated application of small doses of roentgen ray in nonsuppurative tuberculous lymphadenitis. For caseous or suppurative glands, he advises the use of stronger dosage, combined with puncture or aspiration of the liquefied material. He affirms that the roentgen rays probably do not kill the tubercle bacillus, but that they may attenuate its virulence or produce an antitoxin. He employs mild roentgenotherapy in acute infections of the lymph glands. Additional swelling and pain may follow the treatment, but these are transient. The glands rapidly shrink to normal size. In leukemia, Hodgkin's disease and lymphosarcoma, the local and general effects of roentgen-ray treatment are encouraging, but a fatal outcome cannot be prevented. The author cautions against heavy doses in these cases.

Tuberculosis of the bones and joints with or without mixed infection, and in particular the torpid type, responds well to the roentgen ray. Here, too, light doses suffice. Pain disappears, the secretions become serous, and fistulas close rapidly. Too heavy dosage may injure the growth of bones. Leukemic and neoplastic diseases of the bones are also suitable for roentgenotherapy.

OLMSTEAD, Seattle.

A CASE OF METALDEHYDE POISONING. HAROLD BELFRAGE, Acta Pædiat. **6**:481, 1927.

The poisoning occurred from the oral ingestion of "Metatablets," a proprietary Swiss preparation containing metaldehyde, a polymer of acetaldehyde. The immediate symptoms were diarrhea and vomiting, the odor of acetaldehyde being easily recognizable in the vomitus and on the breath. Nervous symptoms developed within twenty-four hours; they were hiccups, an unsteady gait, slurring speech, restlessness. Albumin appeared in the urine. The symptoms gradually subsided, the child making a complete recovery.

HOLT, Baltimore.

Society Transactions

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE.

Dec. 8, 1927

WILLIAM ROPES MAY, M.D., *President, in the Chair*

AURICULAR FIBRILLATION IN CHILDREN. ITS RELATIONSHIP TO RHEUMATIC HEART DISEASE. DR. SIDNEY P. SCHWARTZ and DR. MORRIS M. WEISS.

Auricular fibrillation in children is not as rare as a survey of the literature would seem to indicate. In the children's service at the Montefiore Hospital, fourteen of a total of seventy-one children with chronic rheumatic heart disease showed this type of irregularity at some time during their stay in the hospital. This was 5 per cent of all patients showing auricular fibrillation from all causes seen at the hospital during the same period of time. Fibrillation occurred in 6 per cent of the children at the height of an exacerbation of rheumatic fever.

Clinically, the cases may be divided into three groups: (*a*) the paroxysmal type; (*b*) the terminal type, and (*c*) the chronic or permanent form. In the group studied, one case was of the proxysmal type, four were terminal and the remaining nine were of the permanent form.

The discovery of auricular fibrillation in children is dependent on the care with which the heart rhythm is studied. The development of the arrhythmia seems to be associated with (*a*) active rheumatic carditis, (*b*) mitral stenosis and (*c*) the administration of digitalis in excessive amounts.

In differential diagnosis, this irregularity has to be distinguished from (*a*) a marked sinus arrhythmia which is common in children; (*b*) the irregularity caused by auricular and ventricular premature beats; (*c*) the dropped beat phenomena due to partial heart block, and (*d*) the various tachycardias. The electrocardiogram is of invaluable aid in the diagnosis.

The paroxysmal and terminal types of auricular fibrillation do not admit of any rational therapy.

In patients with permanent auricular fibrillation the problem is to maintain the circulation and to prevent breathlessness, decompensation and the accumulation of fluid. This is done with rest and adequate digitalization. The restriction of fluids and the judicious use of sodium mercurisalicylallylamido acetate in 1 cc. doses repeated at twenty-four hours is of great help in preventing the accumulation of fluid.

Despite the use of these methods, the prognosis is grave. Of our series of fourteen children, six died within one week of the onset of fibrillation, three died within one year, one lived twenty months, another four years and another has reached the adult stage, but he is greatly handicapped by cardiac disease.

DISCUSSION

DR. CARY EGGLESTON: There are so many points which might be discussed that I must limit my remarks to a few which I desire especially to emphasize. The difficulty in diagnosis without electrocardiographic records deserves special mention, and I would stress the comments that have already been made on this subject by the authors.

Perhaps the most striking fact brought out in the present series of cases is that fourteen of seventy-one children, or 20 per cent of all those observed with rheumatic heart disease, were found at some time or other to have

auricular fibrillation. This is obviously contrary to the general conception as to its incidence in childhood. The bad prognosis seems most reasonably referable to the fact that this condition generally occurs as one of the late manifestations of chronic rheumatic heart disease. To me, the same fact seems to suggest an explanation of the possible cause of fibrillation in childhood; namely, the advanced pathologic changes which have taken place in the overdistended auricles. But since uniform structural changes have not yet been demonstrated to account for the development of fibrillation, one must possibly assume, as others have already done, that subtle physicochemical factors may be responsible, presumably changes in the hydrogen ion concentration secondary to minute vascular changes.

The relationship between digitalis intoxication and auricular fibrillation does not seem to me to be quite as clear from the present records as has been suggested. The paroxysmal cases may be dismissed, since it is almost impossible to establish any definite causative relations among them. The first patient in the group with permanent fibrillation seems to have developed the fibrillation at a time when the digitalis intoxication, if present, should have been much less than before and the abnormal rhythm passed off in spite of the continuation of digitalis, relationships which are scarcely in harmony with the belief that the fibrillation was produced by the drug. Another patient who had been taking large doses for long periods did not take digitalis at all for the two days preceding the onset of fibrillation. In this patient, too, the fibrillation seems to have been a terminate event, being followed by death at the end of four days. Some of the remaining cases appear to me also equally difficult to ascribe to intoxication by this drug. I have raised these criticisms merely to question whether or not one is justified in thus incriminating one of the most useful therapeutic agents in the absence of more definitely conclusive evidence.

It might further be pointed out that when one seeks to convert flutter into the closely related fibrillation, it is commonly necessary to administer large doses of digitalis, often such as greatly exceed the known approximate average that is required for maximal therapeutic effects.

Urobilinuria in Children with Heart Disease. Dr. M. H. Edelman.

I wish to call attention to the increased output of urobilin in the urine in the presence of certain diseases and alterations of body conditions. This report concerns itself with urobilinuria in heart disease of children.

Using the method of Elman and McMasters for estimations of urobilin, I have found that quantities of less than 3 mg. per hundred cubic centimeters are normal and quantities of 3 mg. or more per hundred cubic centimeters are pathologic.

I should like to call attention to the fact that cardiac decompensation results in passive congestion of the liver, with consequent hepatic dysfunction and resultant urobilinuria. I estimate the severity of decompensation by the amount of urobilin in the urine at any given time in the course of the disease. I am also enabled by these estimations to gage the amount of recovery at any period, and guide myself in the subsequent management of the affected child. This is substantiated by the increased urobilinuria and the simultaneously poor clinical state which resulted from getting the child with cardiac disease out of bed when he had urobilinuria even though the pulse, temperature and leukocyte count were normal. Urobilinuria also resulted from operative procedures instituted before a return to normal levels.

In 584 reports obtained from children admitted to the wards and cardiac clinic of the pediatric outpatient department of the New York Post-Graduate Hospital, the cases were divided into four groups as follows:

1. Control group (350 patients admitted for diseases of the tonsils and adenoids). 2. Comparative group (32 noncardiac hospital cases). 3. Ambula-

tory group (152 cases). 4. Decompensated group (5 bed-ridden patients with cardiac disease).

The greatest number of urobilinurias occurred in the decompensated group (88 per cent); next, in the ambulatory group (26 per cent), and least in the control group (4 per cent). Urobilin reactions occurred in twelve or fourteen children in the decompensated group subjected to operations such as tonsillectomy, transfusion, extraction of teeth, myringotomy and bronchoscopy. In fifty cases of the control group, only one postoperative urobilinuria was observed and reactions were not seen in five cases of the ambulatory group.

Coincidental complications such as gastric upsets and infections of the upper respiratory tract in the course of heart disease also caused increasing urobilinuria.

The estimations of urobilin are important in the management and care of the child with cardiac disease.

DISCUSSION

Dr. Roger H. Dennett: How this procedure will work out in adults remains to be seen. I have had the opportunity during the summer of watching a large proportion of these fifty children with uncompensated heart disease, and I trust that the rest of the members of the staff were convinced that we received more information from our readings of urobilin than from any other one separate source of information.

First, the procedure is practical. Second, it is simple. The urobilin test can be done in from fifteen to twenty minutes, and twenty tests can be done as easily as one, as is true of many other laboratory procedures. We watched these urobilin charts, which were at the foot of the beds, and when we saw that the urobilin had increased we knew that failing compensation was beginning, even before other signs gave the warning. When one sees the chart of a patient convalescing from scarlet fever, which shows an elevation in temperature, one at once concludes that some infection has occurred in the joints or glands, or that some other complication is starting. In the same way, as we walked through the wards to observe these patients with cardiac disease, a rise on the urobilin chart warned us of failing compensation. We were all extremely interested in the picture of these charts. If a child was allowed to get up too soon, the urobilin went up; the rise in urobilin would be accompanied by other signs of failing compensation, so that we learned to observe the urobilin output carefully. The whole procedure is practical it seems to me, and I believe that those who hear this paper or read it later will use this practical measure.

Papilloma of Esophagus. Report of a Case. Dr. F. Elmer Johnson.

History.—The patient entered the Babies' Hospital, Aug. 22, 1923, at 3 years of age, with a complaint of "vomiting, distension, eructation of gas and constipation." The child was breast fed for five weeks, but did so poorly that she was taken off the breast and given dilutions of grade "B" milk. On account of vomiting, she had had little solid food. Fruit juices were not given at any time, but vegetables were occasionally taken. Since birth, she had required a cathartic or an enema every day.

Vomiting began when the child was 9 months of age and occurred at least once a day for the following nine months. She then had a period of six months without symptoms as long as solids were avoided. She refused food and vomited mucus for one day in her twenty-fourth month; one day in her twenty-eighth month; nearly every day for two weeks in her thirtieth month; every day for two weeks in her thirty-fourth month; every day for two weeks in her thirty-fifth month and every day for the week preceding admission to the hospital.

For six months before admission, she had had periods of swallowing air with subsequent eructation.

Since she was 9 months of age, she had had attacks of pain in the epigastrium lasting from a few minutes to a whole day. These attacks followed the taking of solid food. When she was 28 months old, an attack began at noon and lasted until 4 the next morning, when the pain suddenly ceased. She refused all nourishment during the attack, but when the pain disappeared she was greatly relieved and asked for food. During the attack, she continually "belched gas" and vomited mucus.

At no time had she swallowed an escharotic.

She had had three attacks of pain in the right knee, at 9 months, 24 months and 33 months of age, respectively. Swelling or discoloration were not present at these times, but the mother thought, nevertheless, that the knee had been injured. At the same time the gums were red and swollen.

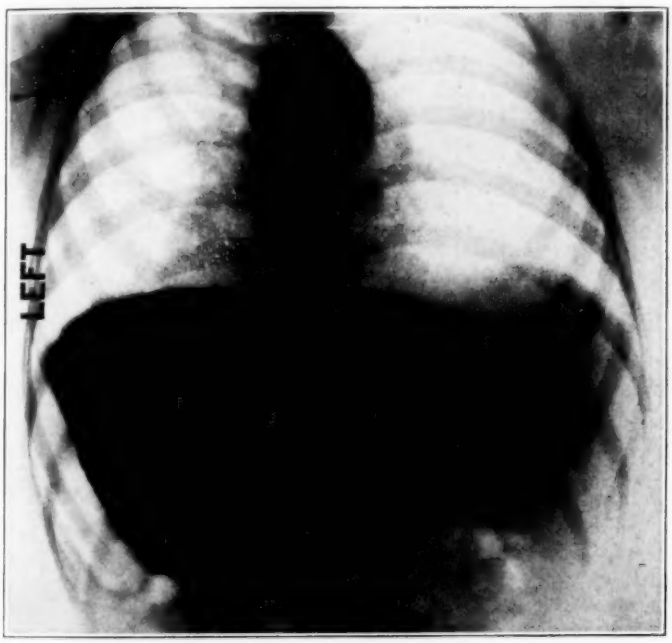

Fig. 1.—Roentgenographic appearance of abdomen in April, 1925, showing evidence of spasm with dilatation of the esophagus above the spasm.

Examination.—On admission, the child weighed 16¼ pounds (7,386 Gm.), and was 34¼ inches (87 cm.) long. She was so emaciated and so feeble that she could not walk. Her intelligence seemed normal. The gums were spongy and congested and bled easily. There were no signs of scorbutus in the long bones, but the previous attacks and the present condition suggested it. The inadequate diet made this seem probable.

She stayed in the hospital 4½ months, until Jan. 5, 1924. The progress in the hospital may be summarized by the house officer's discharge note: "This child has been very interesting and instructive. Since admission, she has gained 3½ pounds (1,519 Gm.) and in general appearance, color and ability to walk has shown much improvement. She could not walk when she entered, but now can run. For the first week in the hospital she had nutri-

tional edema. She vomited only occasionally and appeared to vomit from no apparent cause. Any form of punishment would stop the vomiting for a few days. She is intelligent but nervous. She has been well trained in the hospital, but appears to be the type who would be easily spoiled at home." The regular house diet, except milk, was given until November 11. Thereafter, lactic acid milk, 2 per cent, was given at each meal. Lavage was frequently done without difficulty, and large amounts of mucus were removed.

A series of roentgenograms of the stomach was made on October 27, and the condition was pronounced entirely normal.

She was discharged from the hospital with the diagnoses of "constitutional psychopathy," "malnutrition" and "chronic intestinal indigestion."

Although after leaving the hospital the child improved in general condition, she continued to have the spells of vomiting. In the fifteen months following discharge from the hospital, she gained nine pounds (4,090 Gm.). In April, 1925, another series of roentgenograms of the stomach was done which showed evidence of spasm with dilatation of the esophagus above the spasm (figs. 1 and 2).

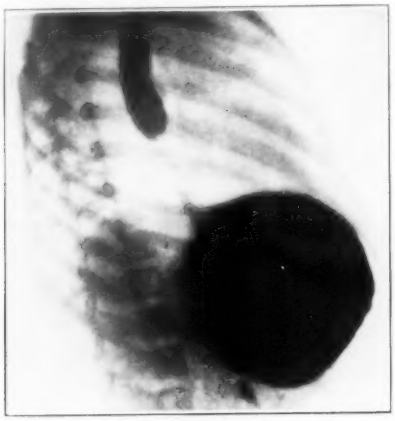

Fig. 2.—Lateral view of condition shown in figure 1.

On account of the roentgen-ray examination, esophagoscopy was recommended. The mother delayed for nearly two months because the child's general condition improved. In May, 1925, however, Dr. John D. Kernan passed an endoscope and found a small tumor 20 cm. from the teeth involving the anterior and left lateral walls of the esophagus. Two weeks later a section 4 by 4 mm. was taken. Histologic examination by Dr. Martha Wollstein revealed the tumor to be a papilloma.

Further surgical intervention has been refused by the parents. When last seen, June 1, 1927, the child weighed 36¾ pounds (16.7 Kg.) and measured 41⅞ inches (106.3 cm.) in length. She does not show symptoms if the food is prepared as gruels and soups. If she eats dry bread or eats rapidly, there is pain.

This child's case is reported because of the rarity of the condition (only three cases in the literature) and because it illustrates the difficulties of diagnosis. Although the child was under the observation of many careful physicians, the diagnosis was not made for a long time.

[NOTE.—The surgical aspects of this case were presented by Dr. John D. Kernan, Laryngoscope **37**:62 (Jan.) 1927.]

ROCHESTER PEDIATRIC SOCIETY AND CENTRAL NEW YORK PEDIATRIC CLUB

Joint Meeting, Nov. 19, 1927

PAUL W. BEAVEN, M.D., *in the Chair*

ORTHOPEDIC TREATMENT OF ACUTE STAGE OF ANTERIOR POLIOMYELITIS. R. R. FITCH.

The matter which I wish to present concerns the prevention of deformity and the restoration of the affected portions of the body to as nearly normal function as possible.

Acute anterior poliomyelitis is due to a virus concerning which little is known. It causes acute constitutional symptoms, and has a selective action on the anterior horns of the spinal cord. A small portion, several segments or the entire length of the cord may be involved. The amount of paralysis depends on the extent of the involvement of the cord. Whether the paralysis is permanent or temporary depends on whether the motor cells of the cord are actually destroyed by the disease, or are merely rendered functionless by the inflammatory reaction associated with the disease process. When motor cells are impaired, muscles dependent on them for innervation become paralyzed. These helpless muscles then become the prey of their functional antagonists or of gravity and are overstretched. Since they do not have power to contract, the lengthening becomes permanent, and the unopposed, unparalyzed muscles contrast, and deformity results.

It seems obvious that treatment should aim to prevent stretching of these weakened muscles. Few, I think, will disagree with this principle of treatment. The question at issue is — when shall this form of treatment be begun? Some contend that it should not be started until after the lapse of a considerable period of time. I believe that it should be instituted as soon as the paralysis appears.

The prevention of deformity must be accomplished by recumbency and mechanical means. Recumbency should be used in cases in which muscles of the trunk are involved. Braces or plaster of paris casts should be used when the paralysis involves the extremities. I usually employ plaster of paris pending the time that braces can be made. The apparatus, whatever it may be, is designed to hold the affected extremity in a position that will guard against the stretching of the paralyzed muscles. If muscles are permitted to be stretched, they become permanently elongated, and consequently can never function normally, even though their nerve supply should be restored during the course of convalescence.

THE KETOGENIC DIET IN THE TREATMENT OF IDIOPATHIC EPILEPSY IN CHILDREN. IRVINE MCQUARRIE.

The ketogenic diet was used in a series of cases of idiopathic epilepsy with favorable effects in the majority. The relationship between the degree of ketosis produced and the freedom from attacks was shown by frequent determinations of the blood and urinary acetone bodies.

Convulsions were induced in patients with severe epilepsy who were benefiting from the ketogenic diet by the administration of large amounts of carbohydrates or sodium bicarbonate. Since blood and urinary acetone bodies are greatly diminished after ingestion of carbohydrates but increased by the alkali given, the tentative conclusion drawn was that the value of the diet is dependent on its acidogenic property.

Methods of calculating and administering the ketogenic diet were outlined.

RAT-BITE FEVER: CAUSATIVE ORGANISM AND ITS USE IN TREATMENT OF PARESIS. STANHOPE BAYNE-JONES.

Spirochaeta morsus-muris was obtained from the skin lesions of a man in Rochester, New York, suffering from rat-bite fever. This is the fourth case in North America from which the organism has been obtained. The spirochete was not cultivated, but was maintained in the laboratory in guinea-pigs and mice. When injected into a man with general paralysis, the spirochete produced typical rat-bite fever, with florid skin lesions. Bayne-Jones at present offered an opinion concerning the effect on general paralysis. The experimental disease can be easily controlled by arsphenamine, but the lesion at the site of inoculation and the skin eruption, which last for some time, are unpleasant factors that must be taken into account in considering the application of this form of therapy.

ROENTGEN-RAY OBSERVATIONS IN ASTHMATIC CHILDREN. S. S. BULLEN and FORREST MERRILL.

A study of the roentgenograms of the chests of ninety-six patients suffering from asthma is presented. Of these, forty-seven are females, and forty-nine are males. Twenty-seven gave positive skin reactions, and sixty-nine did not.

About one-half had their first symptoms during childhood; the remainder developed them during adult life. Forty-five per cent showed roentgen-ray markings within normal limits. The proportion was the same in both the sensitive and the nonsensitive patients. Forty-three of the nonsensitive and four of the sensitive patients had their first asthmatic attacks following long continued infections of the respiratory tract.

The abnormal roentgen-ray observations are classified under the following headings: shadows indicating the result of chronic infection at the bases, thirteen; increased peribronchial markings, eight; evidence of infiltration in the upper half of the chest, fifteen; enlarged hilum, six; emphysema, two; enlarged heart, two, and tuberculosis, four. Two patients showed shadows which were interpreted as areas of absorption atelectasis.

ANTITOXIN TREATMENT OF ERYSIPELAS IN INFANTS. KONRAD E. BIRKHAUG, M.D., Rochester, New York. (From the Department of Bacteriology, University of Rochester School of Medicine and Dentistry.)

During the past two years, in association with physicians in Rochester and with the department of pediatrics of the Strong Memorial Hospital, I have observed the results of the use of erysipelas streptococcus antitoxin in the treatment of thirty-six infants suffering from erysipelas. The ages of these children ranged from 2 weeks to 2 years. These infants presented both the facial and the corporal types of erysipelas. The results obtained with the treatment with antitoxin in infants run parallel with those observed and reported in adults. If adequate doses of the antitoxin are administered during the first three days of the disease, recovery is prompt. If treatment is delayed until complications have occurred, the result is not satisfactory. The average dose of antitoxin in moderately severe cases is from 10,000 to 15,000 units given intramuscularly (from two to three doses of the commercial concentrated erysipelas antitoxin). In extremely severe cases, as many as 30,000 or 40,000 units were necessary in order to induce a crisis in temperature and pulse. The intramuscular route is preferable unless the infant is dying, in which case the intravenous method of administration should be employed. Repeated injections of 5,000 units every eight or twelve hours should be given until the temperature drops to within normal limits and remains sustained. Among the thirty-six infants specifically treated with the antitoxin, five died; intercurrent streptococcic peritonitis, septicemia and bronchopneumonia complicated three of these

deaths and the remaining two infants died with frank facial and pectoral erysipelas. The days of admission of these patients were, respectively, the third, fifth, seventh, seventh and thirteenth day after the onset of the disease. Considering the extremely high mortality among infants with erysipelas under 2 years of age, it is safe to say that the streptococcic antitoxin for treatment of patients with erysipelas has changed the grave prognosis, the results obtained being commensurate with those observed in the treatment of diphtheria.

BEHAVIOR PROBLEMS IN CHILDREN. ERIC KENT CLARKE, M.D. ..

The time honored conception of the psychiatrist is that he deals only with people who are insane, and consequently he is a man to be avoided. This fear on the part of the general public, and unfortunately an idea that is carried over to the general practitioner, has done much to retard the development of psychiatry, particularly in its relation to work among children.

In most of the children referred, behavior problems have reached the stage of real anxiety, fear for future on the part of parents and school authorities. A study of them reveals problems in personality which originate in unsatisfactory adjustments in the home, poor school placements and environmental factors that produce unruly episodes in an otherwise normal child. Today it is considered well worth while to spend as much time on these everyday manifestations as on children who are not definitely abnormal. Abnormal children are fairly frequent visitors to our clinics; the majority are normally endowed, but they have not made the right adjustment, so that they cannot keep time with the general reaction of the rest of group.

In the case of three children one illustrated a maladjustment in the home. A boy, normal mentally, was of the type who had tantrums and otherwise produced "scenes." A second case illustrated a school problem. A mentally inferior Italian girl had a sick-complex the operation of which enabled her to miss school work which she disliked. A third child was typical of a clash with community interest. A bright boy stole and indulged in sex perversion with the "gang." In each case, an adjustment was made more or less successfully, and suggestions for further treatment were outlined.

FLEXNER'S DYSENTERY WITH MULTIPLE PERIPHERAL PARALYSES. S. W. CLAUSEN.

Two sisters, aged 5 and 7 years, were attacked by dysentery of the Flexner type in August. Both became speechless and developed bilateral wrist drop and toe drop. The child whose illness was more severe remained speechless for ten weeks and showed marked ataxia during convalescence. Both children have practically completely recovered. The cases are of interest because of the rarity of such nervous symptoms in this type of dysentery.

AMEBIC DYSENTERY WITH MOTION PICTURE DEMONSTRATION OF AMEBAS. J. M. PARKER.

An Italian boy, aged 4, who had never been away from Rochester developed diarrhea, melena, colic and a low grade fever during September, 1926; repeated cultures did not show pathogenic bacteria in the stools. A foreign body was passed in the stool on October 3, and the patient improved somewhat under treatment with tannic acid and enemas. Proctoscopic examination repeatedly revealed hyperemic mucosa with ulcers the size of a pin point. Examination of scrapings on the warm stage finally revealed ameba histolytica. After a routine course of treatment with iodo-oxyquinolin sulphonic acid, the amebas permanently disappeared.

LYMPHOID APPENDICITIS. PAUL W. BEAVEN.

Janet C., aged 5, suffered with pyuria of two and one-half years duration and intermittent abdominal pain for seven months. Only temporary relief

followed treatment with alkalies, urotropine, and hexylresorcinol. Two weeks before operation, colicky pain became almost constant about the umbilicus, fever developed and the patient began to vomit practically everything. The abdomen was soft and free from tenderness. Laparotomy revealed an appendix which seemed grossly normal, but which, on section, showed lymphoid hyperplasia and moderate infiltration by leukocytes. The child has had neither pyuria nor any complaint for the two years following the operation. Two other similar cases have been observed.

RETROBULBAR NEURITIS. HERBERT C. SOULE.

A case of sudden blindness without other symptoms occurred in a child, aged 3. The family and past histories were negative. Positive physical observations were: fixed pupils, right disk slightly hazy and left disk pale. Positive laboratory observations were: tuberculin test positive, roentgenogram of antrum suggested involvement, but this was not confirmed by direct examination. The diagnosis was obscure. Many possibilities were considered as tumor of the brain, encephalitis, meningitis, retrobulbar neuritis, wood alcohol poisoning and others. With her improvement in the hospital and other negative observations, retrobulbar neurities seemed most likely. The etiology was unknown. The observations were the same at Johns Hopkins Hospital except the evidence of sinus trouble was not found. The patient has been on tuberculin treatments the last eight months, and her vision has improved considerably; both disks are still slightly pale. A brief review of tuberculin treatment in conditions of the eye is given. The patient was presented.

POISONING FROM SHOE-DYE. JOHN AIKMAN.

Three cases of poisoning from shoe dye are reported in children aged 6, 13 and 9½, respectively. The symptoms were marked cyanosis, general weakness, headache, nausea, vertigo and prostration. One child had a convulsion and became unconscious. The exact toxic agent is not known, but both aniline and nitrobenzol, used as solvents for the pigment, are reported as the cause. Most patients recover after a few hours, but two deaths were recorded in the sixty-one cases previously reported.

HEMORRHAGE IN SUPRARENALS IN A CASE OF STATUS LYMPHATICUS. E. R. REYNOLDS.

A case was reported with a history of sudden death with symptoms of abdominal disturbance followed by different respiration and later by shock. Complete autopsy showed: hyperplasia of thymus and of the lymphoid system; bilateral hemorrhage in the suprarenals. Because of the age of the patient (3 years) the following conclusions were made: abnormal physiologic changes of the glands produced hypofunction of the suprarenals, resulting in hyperplasia of the thymus and other lymphatics with (*a*) diminished sympathicotonia and (*b*) vagotonic irritability. Vascularity of the suprarenals may aid in the production of hemorrhage produced through an exaggeration of the vagotonic state.

THROMBOPENIA. DAVID WOLIN.

A girl, aged 9, who had undergone uncomplicated tonsillectomy at the age of 7½ suddenly developed extensive purpura and epistaxis. The blood platelets numbered 14,200. Capillaries were ruptured below the tourniquet, and direct transfusion was made. Three weeks later epigastric pain, hematemesis and melena, leukopenia and profound anemia developed. The red blood cells numbered 1,600,000, and the platelets, 11,600. Six transfusions and quartz lamp

therapy did not result in amelioration of symptoms or in an increase in the number of red blood cells, white blood cells or platelets. Later transfusions were followed by severe reactions suggestive of anaphylaxis. The patient's condition did not permit splenectomy. Death occurred after forty-one days. There was frequent unexplained fever to 104 F.

INVOLUNTARY MOVEMENTS IN FOUR PATIENTS. R. S. LYMAN and D. M. RIOCH.

Moving pictures were shown in the following cases to illustrate the movements:

Dystonia Musculorum (case 1): Rigidity was evident in practically all muscles, except those of the head and face. There was extreme resistance to passive manipulation, suddenly relaxing to recur again. The feet were shortened by contractures, especially of the extensor muscles. There was a tendency toward frequent pronation of the arms, and a suggestion of associated movements. Neither oscillation nor true tremor was present. The movements appeared to be purely the result of ever-changing muscular spasms.

Chorea (case 2): Typical irregular jerky movements were shown in the arms. Muscular rigidity was not present, and there was no real tremor. With slow movements, the choreic twitches sometimes resembled athetosis. Athetosis was the only involuntary movement which Gordon Holmes found consistently associated with lesions in the basal ganglia. Choreic movements often followed lesions in that region but were not infrequently assigned to lesions elsewhere.

Hereditary Systemic Degeneration (case 3): A similar disorder through at least three generations was revealed in the family history. Slight diffuse rigidity was present. Marked tremor, which was regular in rate and amplitude and which was increased by action and emotions, involved several different physiologic muscle groups at the same time, being most marked in the hands and fingers and was not coarsely magnified on approaching the goal (not intention-tremor). Slow movements showed progressive waves of motion, when the patient reached for a glass of water; with the unaided eye, the hand appeared to hit the glass with a single movement overshooting the mark. Paresis of all extra-ocular movements was present, and there was psychomotor slowing. Tremor, not rigidity, was the outstanding feature.

Postencephalitis Parkinsonism (case 4): Marked diffuse rigidity was evident. Tremor was not as extreme in excursion but was otherwise similar to that in the preceding case. Parkinsonian gait, drooling and other symptoms were present. Rigidity rather than tremor was predominant. Walshe showed that rigidity can be temporarily abolished with procaine in such cases, without affecting the tremor in any way and without destroying the voluntary power over the limbs.

These pictures were intended to emphasize certain well known advantages in recording motor phenomena with the moving pictures, both for immediate study and for future reference in the comparison of changes in the course of a disease. All four patients appeared to have motor disorders of a type usually attributed to lesions of the basal ganglia. They were selected in order to contrast in particular the rôle of "rigidity" and "tremors"; the latter are relatively more common, variable and uncertain in their localizing value. Dystonic spasm, choreic twitches and tremor with more or less diffuse hypertonicity of muscles were shown. A similarity was pointed out in the superficial appearance of a case of postencephalitis (rigidity and tremor; pathologic lesions usually considered limited to parts of the basal ganglia) and a case of hereditary systemic degeneration (rigidity and tremor of the same type; pathologic lesions notoriously scattered through cortex, basal ganglia, midbrain, cerebellar system and cord). It is felt that attempts at definite anatomic localization of lesions from the appearance of patients with rigidity and involuntary movements are

less to the point than is a physiologic approach which aims at clearer ideas of functional relationships and at treatment in cases of rigidity and of tremor possibly as separate mechanisms.

SKIN REACTION IN RHEUMATIC FEVER. ALBERT D. KAISER, M.D.

Studies on the production of soluble toxins produced by *Streptococcus scarlatinae* and *Streptococcus erysipelatis* suggested to Dr. Conrad E. Birkhaug a study of possible toxin production by a series of other streptococci of the hemolytic and nonhemolytic types.

From a girl, aged 5, who had had rheumatic fever and endocarditis, Birkhaug obtained a nonmethemoglobin-forming streptococcus in the blood culture and a similar strain from the mitral valve vegetations at autopsy. This strain was found to produce a potent, soluble and thermostable exotoxin, 1 cc. of an eight day culture containing more than 1,000 skin test doses of the toxin.

Utilizing the filtrate of the eight day culture of the nonmethemoglobin-forming streptococcus isolated from patients with rheumatic fever, Birkhaug made skin tests similar to the Schick and the Dick tests with 0.1 cc. of a 1:100 dilution of the toxin in normal physiologic sodium chloride solution, to which had been added 0.5 per cent phenol. Control tests were made with the same toxic filtrate subjected to 100 C. for more than one hour. Birkhaug noted that the skin reactions with the filtrates from cultures of *Streptococcus scarlatinae* and *Streptococcus erysipelatis* (hemolytic streptococci) as well as *Streptococcus viridans* were positive in the same percentage in normal persons and in persons with definite histories of rheumatic fever. On the other hand, when normal persons were tested with filtrates from the cultures of the nonmethemoglobin-forming streptococcus isolated from patients with rheumatic fever, it was found that about 15 per cent reacted positively, while in those with manifestations of rheumatic fever, about 70 per cent reacted positively. These results suggested a possible relationship between the toxin produced by the nonmethemoglobin-forming streptococcus of Birkhaug and the hypersensitive state of the skin of persons stigmatized by rheumatic infection.

Children were tested on the flexor surface of the forearm in a manner similar to that employed in making the Schick test, A solution of from 1 to 100 was used, and for the first tests a control was made. The control tests, however, were consistently negative; so they were subsequently discontinued. The result of the test was read in twenty-four hours. Reactions were present earlier, but the maximum redness was present at twenty-four hours, after which period it would disappear rapidly. In most instances the positive reactions produced an erythema about 2 cm. in diameter, similar to that produced by a positive Dick test. In the strongly positive reactions a small erythematous nodule would appear at the site of the injection, as described by Birkhaug in his original publication.

Summary of Tests.—1. Eight hundred children from 1 week to 16 years of age were tested intradermally with 0.1 cc. of a 1:100 dilution of the toxic filtrate from cultures of the nonmethemoglobin-forming streptococcus isolated by Birkhaug from cases of rheumatic fever.

2. Approximately 32 per cent of all the children reacted positively to the test.

3. Separating children into groups, it was found that the reaction was positive in 20 per cent when there was no history of rheumatic infection or repeated attacks of sore throat; in 35 per cent when there was a history of repeated attacks of sore throat, and in 72 per cent when there was a history of rheumatic infection.

4. There is no reaction parallel to that obtained with the Schick or the Dick tests.

CHICAGO PEDIATRIC SOCIETY

Clinical Meeting, Children's Memorial Hospital, Nov. 14, 1927

JOSEPH BRENNEMANN, M.D., *Presiding*

VERTEBRAL DISEASE WITH MENINGEAL SYMPTOMS. Presented by DR. E. J. BERKHEISER.

I want to show you this boy, who is about 10 years of age, because of the difficulty I had in making a diagnosis, and also to show the importance of diagnosis in his condition. He was admitted to the hospital on June 3, 1927, with the history from Dr. Mayers that on March 25, 1927, he had had an attack of meningitis. The spinal fluid was under increased pressure and contained 1,500 cells. A culture was negative for meningococcus, but smears showed a few other organisms. He was sent to the Municipal Contagious Disease Hospital, where he was given serum treatment and gradually improved. He was discharged on May 3, and in forty-eight hours had a relapse, with severe headache and vomiting. He was returned to the hospital, where cultures were again negative, but the cell count remained high. The Wassermann reaction on the spinal fluid was 2+ and on another occasion 4+, though the blood Wassermann reaction was negative. The patient improved under treatment, but some rigidity of the spine persisted, and because of that he was sent here. There was nothing of importance in his history except the attacks of meningitis, and the fact that in February, one month before his first illness, he had a boil on the back of the neck which was not opened.

Examination of the back showed an extreme rigidity of the entire vertebral column, up to the cervical region. The patient lay in bed and stood with marked lordosis, and bent over as if he might fall when trying to walk. There was no special deformity of the back in the lumbar region, and no indication of any special pathologic change in that region, but because of the stiffness of the back due to muscle spasm I asked repeatedly for roentgenologic examination. Not until June 27 was I able to obtain a picture which showed anything which I considered pathologic.

Under treatment the condition of the spine improved, the back became flexible, and on September 2, the patient was discharged from the hospital. He had been lying on a Bradford frame, and the back was immobilized in a plaster cast when he was discharged. He went to school, going back and forth in one of the large school busses, and the shaking made him complain again of pain in the back. On November 11, he had headache and epigastric pain. A blood count showed 15,600 leukocytes. The temperature was 102.6 F., and there was definite stiffness of the neck. Today he is much better, and the Kernig sign is less marked.

In this case there is a possibility of several conditions. From the roentgenograms of his back it was at first thought that he had Pott's disease, because this is the common pathologic condition in children, but I do not believe that the process is tuberculous. Lateral dislocation or the proliferation of bone shown in these pictures is not encountered in Pott's disease. There should be a greater amount of destruction in the vertebrae by this time if it were Pott's disease. There is no history of typhoid fever, and I have not thought of the possibility of the spine being affected by typhoid. The possibility of pyogenic involvement of the vertebrae must be considered. The condition may be syphilitic meningitis and a syphilitic process of the bones of the back. Perhaps I shall give him a rather strenuous course of antisyphilitic treatment. It would be hazardous to perform a spinal puncture in this region. I should like to have any suggestions regarding the treatment or diagnosis.

DISCUSSION

DR. ARCHIBALD HOYNE: Is is possible that the spinal punctures have had anything to do with the trouble?

DR. BERKHEISER: I think that is a possibility, because such things do happen. When repeated spinal punctures are made, there may be an injury to the intervertebral cartilage.

DR. HOYNE: I do not think the child had epidemic meningitis. He had some trouble with his back, but he did not have any ordinary symptoms of meningitis involving the brain. He looked as though he had some kind of process in the spine that was discharging pus into the spinal canal. There was no evidence of anything abnormal in the cranium.

DR. BERKHEISER: I think it was rather a meningismus. The cells ranged from 550 to 2,200 and were as numerous as 15,000.

CONGENITAL ABSENCE OF FEMUR. Presented by DR. F. SEIDLER.

This is a rather rare deformity, and there are four different degrees of malformation of the femur: (1) The femur is well shaped, but small; (2) the upper end of the femur is misshaped, in the sense of a coxa vara; (3) only separated pieces of the femur are present, but there is a well shaped knee, and (4) there is only a rudimentary piece of femur, in connection with the tibia, without a joint.

This boy is 2½ years of age. He has a well shaped pelvis, and the head of the femur is in the socket, but there is no neck or shaft. There is a knee joint but no patella. The thigh is in right angle flexion and adduction. There is complete absence of the fibula and several metatarsal bones. His heel was displaced upward and backward, and the foot was in extreme dorsal flexion, but by manipulation I succeeded in making the foot straight. The next task will be to construct a brace to enable him to step on his foot and compensate for the difference in length, at the same time giving support to the pelvis.

TREATMENT OF BURNS BY TANNIC ACID. Presented by DR. MONTGOMERY.

Fifteen cases were shown, some by lantern slide and photograph as well as the children themselves, to illustrate the effect of this treatment. In 1925, Davison of Detroit described the treatment of burns by tannic acid, and this method has been followed in the hospital since 1926. According to Davison, a 2.5 per cent tannic acid dressing should be used as soon as possible. This is put on in the form of wet dressing and left for twenty-four hours. At the end of that time coagulation has formed, and the wound is allowed to dry. It forms a dense heavy covering and is usually dry. It is smooth, leathery, dry and insensitive. Dressings are not used when coagulation has formed. The child is put in a tent with an electric light and a sterile sheet to protect the burn. Blebs, if any, are opened. The dressings were uncomfortable; so the spray has been used. The burn is sprayed immediately, and the spraying is repeated in about fifteen or twenty minutes again and again until coagulation has formed. A 5 per cent solution is used, freshly prepared, because it forms gallic acid if allowed to stand. Fluids are pushed, and sometimes a little morphine is used if there is shock. Blood and dextrose transfusions are given at times. In addition to that the child is simply kept at rest. Heretofore sterile petrolatum has been used. This treatment with tannic acid has accomplished several things. In the first place, fluid has not been lost through seepage, which is important. The burn is kept clean, and the covering remains sterile unless there is leakage or unless it is a deep burn. The toxemia is much lessened. The patient does not show so much delirium. There is a marked rise in the urea in the blood for the first twenty-four hours, when it reaches its peak; then it subsides until at the end of four days it is back to normal. The skin is cleaned and sprayed with tannic acid until a firm surface

is obtained. There is no question about its lessening pain; there is a definite analgesia, and the patients suffer much less than under any other form of dressing. It also lessens the toxemia, the loss of fluid and the absorption. In burns of the face Davison advocated ointment made with tannic acid. This was tried, but the spray used carefully was found more effective.

TUBERCULOUS PERITONITIS: TUBERCULOSIS OF THE SUPRARENALS (?). Presented by DR. GEORGE EDWIN BAXTER.

This case is unusual from two standpoints, so far as the present diagnosis of generalized tuberculosis is concerned; first, because of the rather stormy career of the child, and second, because of the unusual color of the skin. The child was rather dark at birth, but this has become much more pronounced during the last six months. The mother's skin is slightly dark, but the father is of the blond type. I do not think the dark color is due to the violet-ray treatment, for during a period of several months when radiotherapy was not used, the skin became even darker.

The child is distinctly anemic, the abdomen is enlarged and there is some fluid present, which was not present a month previously. She was brought to me in July, 1926, with a history of having had bloody stools more or less of the time for six or eight months. At that time the stools consisted chiefly of mucus with a slight amount of blood, and she had five or six such stools a day. She had lost her appetite and considerable weight. She was kept under observation for several months, and she gained about 2 pounds (0.9 Kg.) in weight, the blood disappeared from the stools and she had a general feeling of well being. During the fall of 1926 she developed some disturbance of the upper respiratory tract and lost ground rapidly. This was repeated two or three times during the fall, and in December she was placed in the hospital and kept here for about two months. The hemoglobin had been around 50, but just before she entered the hospital it was down to 23 or 24, with 2,000,000 red cells and 19,000 leukocytes. On Jan. 22, 1927, she was given 200 cc. of whole blood from her mother, by intraperitoneal injection. Following this she was uncomfortable and restless. Then she was given 60 cc. intravenously. She had a marked reaction; the transfusion had to be stopped while she was on the table, and it looked as if she would not survive. She was extremely ill, and about that time showed signs of otitis media, followed by involvement of the mastoid on the right side. The mastoid was operated on in February, 1927, and since then the bowel movements have been practically normal, and there has been little blood present in the stools. She has made a rather satisfactory recovery, except that she has not gained in weight.

Roentgenograms of the chest made at the time of admission were interpreted by Dr. Davis as showing pulmonary tuberculosis. Several intracutaneous injections were made and a Pirquet test, all of which were negative for tuberculosis.

She went home, and during the spring was fairly well, except that she had two or three acute infections of the respiratory tract and lost weight. This fall she returned to the hospital, with the abdominal condition as it is now. There is marked increase in size, and fluid can be demonstrated at this time. She will be 4 years old in January and weighs about 22 pounds (10 Kg.), which is a gain of 1 pound (0.5 Kg.) since her last acute infection, and approximately what she weighed a year ago. The stools have been repeatedly examined for parasites and have always been negative.

It seems to me most unusual for a case to progress over such a period of time with so little general improvement, and not much evidence of retrogression. About two weeks ago, it was thought that she was losing rapidly, but she improved, has gained 1 pound (0.5 Kg.), and is as well, if not better, than before. If the condition is tuberculous, it is possible that there is involvement of the suprarenals. This condition occurs in adult life, but no cases have been reported in which the patients were under 15 years of age. It is

believed that most of the cases are the result of tuberculous infection. If that is true, why is it not possible for tuberculosis of the suprarenals to occur in childhood, and for the resulting symptom-complex to be the same as in Addison's disease which occurs during adult life? All tests for syphilis in this case have been negative.

NEPHRITIS (CHRONIC, GLOMERULAR). Presented by DR. C. A. ALDRICH.

This patient demonstrates that most things are possible in medicine. He was first seen in November, 1925, with a history of acute nephritis of three months' duration, with blood in the urine. He had edema when he entered, but the blood pressure was normal. A Mosenthal test showed moderate restriction in the middle field, from 1,010 to 1,018. The blood chemistry was normal, except for a slight increase in cholesterol. The edema persisted for several months. While in the hospital, the patient had a tooth removed, which was said to have caused trouble at the onset of the disease. He was given gas anesthesia, and pus was found in the right antrum. He was discharged on May 24, 1926, but was kept under observation in the outpatient department. There was marked albumin in the urine, and on Jan. 31, 1927, it was decided that because of this and the pus in the antrum, irrigation of the antrum was justifiable. On the day he entered, to my great astonishment, albumin was not present in the urine. Both antrums were drained, and pus was found. There was a marked reaction. The temperature went up, he developed an otitis media which had to be drained, and blood, casts and albumin appeared in the urine. This attack lasted for one week, since which time there has been nothing abnormal in the urine.

I think the case represents a hemorrhagic nephritis which progressed to a chronic stage. The boy is perfectly well today according to all tests.

NEPHRITIS (PARENCHYMATOUS); NEPHROSIS. Presented by DR. C. A. ALDRICH.

CASE 1.—This child is presented to show the distribution of edema. He shows practically no edema of the upper extremities at present, but two days before presentation, he had a marked edema of the face. He is in the stage of dependent edema. He will have a diuresis in about a week or ten days, because I have seen this happen many times.

CASE 2.—Here is another boy who has just had such an attack. This child gained 50 per cent of his body weight in fluid; there were 18 pounds (8.2 Kg.) of edema fluid in a boy who weighed 34 pounds (15.4 Kg.). He went through the same course as the boy in case 1 and has become practically free from the edema, though there is still albumin in his urine.

CONGENITAL ATRESIA ANI PERINALIS. Presented by DR. JOHN L. REICHERT.

This child presents a history of no bowel movement for the first three days of life, and stools thereafter that were infrequent, diarrheal and resulting only from violent catharsis. She has never vomited. At 2½ months of age her weight is less than that at birth.

The position of the anus is covered with a grayish, wrinkled skin. One half inch anterior to this, there is a fistulous opening in the midline of the perineum admitting only a probe. On straining, the anal region bulges to the size of a small orange, and a thin ribbon stool is squeezed from the fistulous opening.

Gradual dilatation was performed, and in a week the opening admitted the little finger. Obstruction was not found above the opening, but the rectum was markedly pouched. The infant then gained 6 pounds (2.7 Kg.) in two months, and the stools were normal, so that it was unnecessary to perform further dilatation. Recently, however, the opening has become smaller, and further dilatation is necessary.

In simple imperforate anus of this type due to the failure of invagination of the proctodeum, but with complete formation of the rectum, the meconium may burrow to find exit in a variety of locations, as into the urethra, vagina or bladder (atresia ani, urethralis, vaginalis or vesicalis). The perineal location of the opening is the optimum one, since there is no danger of infection of the genito-urinary tract, and surgical intervention can be postponed, normal bowel movement being maintained by periodic dilatation.

INFECTIOUS MONONUCLEOSIS. Presented by DR. JOHN L. REICHERT.

The history of this infant was uneventful until he was 5 weeks of age. At that time, he developed an infection of the upper respiratory tract, with vomiting, marked constipation and a temperature of from 102 to 104 F. During the course of the infection, he went into collapse several times. Each attack was associated with marked gastric distention and was promptly relieved by the passage of a stomach tube. The duration of acute symptoms was five days. The blood picture was as follows: red blood cells, 4,160,000; hemoglobin, 80 per cent, and white blood cells, 15,600. The differential count was: granular leukocytes 18 per cent; small nongranular leukocytes 4 per cent, and large nongranular leukocytes 78 per cent. All cells were normal except the large nongranular cells, which were of a heterogeneous type. They were all large and mononuclear, but with nuclei of widely different size, shape and staining. The blood picture gradually returned to normal in two weeks.

Infectious mononucleosis does not show a definite clinical picture with the exception of the blood picture and an acute infectious process, usually but not always of the upper respiratory tract. It differs from acute lymphatic leukemia by the absence of anemia, the lack of uniformity of the mononuclear cells, the short course and the invariably favorable prognosis.

HEMORRHAGE (SUBPERIOSTEAL) IN INFANTILE SCURVY. Presented by DR. JOSEPH BRENNEMANN.

About a week before admission to the hospital, this child had symptoms of scurvy, with extensive subperiosteal hemorrhage of the lower end of both femurs. In a roentgenogram made two and a half weeks later, the dense shadow of the organized and somewhat calcified areas of hemorrhage was seen. About ten days later, the condition was unchanged. It was then found that the child had vomited all the orange juice that had been given. In the roentgenogram made on April 27, the areas were distinctly smaller on both sides, and after nearly two months, they were much smaller but still evident. About that time, the child left and was not seen again until October 5, when he had a fracture from an insignificant cause. There was practically nothing left of the original condition, the thickening having cleared up completely, but he had blue sclerae.

BLUE SCLERAE AND MULTIPLE FRACTURES. Presented by DR. JOSEPH BRENNEMANN.

This child came into the hospital with a history of being well up to the time of admission, except for a fracture of the arm. She was brought to the hospital because while playing in bed with her little sister she was kicked lightly on the thigh, and a fracture of the femur resulted. This is interesting, because the child has the bluest sclerae I have ever seen. So far as I can ascertain, there is no history of fractures in the father's family. In the mother's family, there were twelve children by the same parents, and she does not know of parental fractures on slight provocation; however, she and one brother, of the twelve children, had blue sclerae, and they alone had multiple fractures on slight provocation; they had such fractures principally before the age of puberty, although she had recently fractured her left ulna, which had been broken four or five times. In childhood, she broke the bones of the lower

part of the leg several times, and believes she dislocated a joint of one finger. One of her brother's fractures was of an arm, but she is unable to state which bones were involved. The patient has one sister, 1½ years old, who has white sclerae and has not had any fractures.

One could hardly say these were typical cases of fragilitas ossium, unless one included under that term all fractures produced by slight causes. Fractures did not occur at birth or shortly afterward. Whether blue sclerae are associated only with typical fragilitas ossium or with other conditions in which bones are easily broken, as in these cases, is hard to determine. That there is such an association is common knowledge, but the explanation for it is not clear. One German observer has said "Here is a faulty anlage," a faulty development of the mesoderm, as a result of which the pigment of the blood and of the blood vessels shows through the ectoderm. I have looked for blue sclerae for a long time and have seen some instances of it, but these are in a class by themselves in my experience, for the color is a deep sky blue.

MULTIPLE NEURITIS. Presented by DR. S. H. KRAFT.

This child is 5 years of age; he entered the hospital three days ago with the complaint that he was having difficulty in walking. The disturbance came on two weeks before and almost from the onset it was as severe as at present. There is no history of any preceding sore throat or acute infection of any type. There has not been any rise in temperature.

The interesting thing about the case is the extreme rarity of multiple neuritis in children. In one series of Abt's textbook, two cases are reported, and in another, over ninety cases, of which about ten occurred when the patients were between the ages of 9 and 19; the condition did not occur in any patient below that age.

This boy shows extreme generalized weakness which involves both the upper and lower extremities and the muscles of the trunk. Both the deep and superficial reflexes are absent. There are no signs of complete paralysis; the speech is clear. Changes have not occurred in the heart or chest; there are a few enlarged glands in the neck and some redness of the throat. A Schick test was performed, but as yet it has not shown anything. Spinal puncture has not been made.

Multiple neuritis is extremely rare when it is not due to diphtheria. In most cases on record, sensory disturbance is reported, but there is none in this case. The child complains of a little pain in his leg and shows a little pain on pressure over the nerve points, which made me feel that the condition might, after all, be due to diphtheria. In diphtheritic neuritis there is nearly always some history of primary sore throat, or some pharyngeal or nasal infection. The only evidence that an earlier sore throat might have been a factor is the enlarged glands in the neck and the reddened tonsils. Against the diagnosis of poliomyelitis is the symmetrical distribution of the involvement. The patient has an internal strabismus which is congenital and has nothing to do with the condition.

BILATERAL THROMBOSIS OF THE FACIAL AND ANGULAR VEINS. Presented by DR. S. C. PEACOCK.

This child was brought into The Children's Memorial Hospital on May 17, 1927, with the complaint that the right ear had been discharging for two months and that there had been a painful swelling behind the same ear for three days. Physical examination revealed a girl, aged 22 months, who was in good condition except for a temperature of 102 F., and the swelling behind the right ear which involved the subcutaneous tissue both behind and in front of the ear. Some fluctuation was present over the mastoid area, and yellow pus oozed out of a sinus in that region. Dr. Livingston performed a simple mastoidectomy ten days later and evacuated some pus from the antrum. He said that the

bony tissue was more spongy than normal. Eight days later, the child had a slight fever, a poor appetite, some swollen glands behind the angle of the left jaw and a mass which had about the consistency of rubber palpable mostly in the left side of the pharynx. Apparently, there was an extensive hemorrhage into the tissues of the face, over an area measuring approximately 11 by 6 cm., and involving both maxillary regions as well as the upper lip and nose as far as the glabella. It extended laterally a little more on the left side than on the right side of the face. This region was bluish black, swollen, tense and insensitive to pain. Four days later, the patient developed some difficulty in breathing and opisthotonus. The mass in the pharynx was fluctuant. A large amount of thick greenish-yellow pus was obtained from this abscess, containing mostly staphylococci and pneumococci. The mastoid wound healed little, if any, and later became infected with *Bacillus diphtheriae*. The margins of this black mass retracted and finally sloughed out in toto after five or six weeks, exposing the maxilla with loose teeth and the turbinate regions. This area became coated with a slimy, gray exudate which was kept fairly clean with wet dressings. Two skin grafts were made, but neither was successful. About twelve hours after the last skin graft, the patient developed a high temperature and died suddenly without any notable physical changes. The abrupt onset apparently precludes the diagnosis of a noma. It has been suggested that this might be a thrombosis of the facial artery, but it would seem from an anatomic standpoint, that one would have to consider this as a thrombosis of both the anterior facial and the angular veins, probably secondary to the retropharyngeal abscess.

Book Reviews

LA TETANIA NELL'INFANZIA. By S. FABRIS. Pp. 180. Naples: Edizioni "La Pediatria," 1927.

Within 160 pages of text and a bibliography of about 500 references, a comprehensive review of the subject of tetany is presented. The author's own observations extend over a period of ten years.

The work is divided systematically into seven sections, each dealing with the various phases of the subject. Each section is further subdivided to discuss its special aspects. The main divisions are as follows: symptomatology, differential diagnosis, pathologic anatomy, etiology, pathogenesis, prognosis and therapeutics.

The section on symptomatology is divided into two parts; the first part is a discussion of manifest tetany, which includes carpopedal spasm, laryngospasm and bronchospasm; the second part discusses latent tetany, which includes the Chvostek's sign, Trousseau's sign, Lust phenomenon, Pool-Schlesinger's sign and other objective observations.

The author does not consider tetany as belonging to the "spasmophilic diathesis" group which includes laryngospasm and eclampsia. He regards laryngospasm as one of the symptoms of manifest tetany and eclampsia as an independent symptom which may occur in tetany or in other diseases.

Twenty-five pages are devoted to a minutely detailed discussion of the pathologic physiology of the disease.

The book could be much shorter and still retain its completeness.

CORRECTION

In the article by Drs. Toomey and Hauver, "Intradermal Vaccination" (AM. J. DIS. CHILD. **35**:186, 1928), an error occurred in the second paragraph of the section "Method and Material," on page 187. The dosage should read "0.1 cubic centimeter [instead of one cubic centimeter] of this 1 to 4 dilution from bulk or capillary vaccine was injected." Also, in Table 1, p. 189, the word "months" in each line should read "weeks."

Directory of Pediatric Societies

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION OF DISEASES OF CHILDREN

Chairman: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Minneapolis. Time: June 11-15, 1928.

AMERICAN PEDIATRIC SOCIETY

President: Dr. J. Claxton Gittings, 1828 Pine St., Philadelphia.
Secretary: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Washington, D. C. Time: April 30 to May 2, 1928.

THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. J. P. Crozier Griffith, 1810 Spruce St., Philadelphia.
Secretary: Dr. Paul W. Emerson, 86 Bay State Road, Boston.
Place: Minneapolis. Time: June 12, 1928.

AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 Seventh Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 Seventh Ave., New York.
Place: Stevens Hotel, Chicago. Time: October 15-19, 1928.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. G. R. Pirie, 20 Madison Ave., Toronto.
Secretary: Dr. A. P. Hart, 1844 Bloor St. West, Toronto.
Place: Vancouver, B. C. Time: June, 1928.

SECTIONAL

CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. H. T. Price, 121 University Place, Pittsburgh.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.

SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

Chairman: Dr. E. C. Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. J. Waring, De Renne Apts., Savannah, Ga.
Place: Asheville, N. C. Time: November 12-15, 1928.

LOCAL

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. W. L. Channell, Medical Bldg., Oakland, Calif.
Secretary: J. E. Parrott, Medico-Dental Bldg., San Francisco.
Place: Various Teaching Schools. Time: Monthly.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary and Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. W. Wiley Jones, Majestic Bldg., Denver.
Secretary: Dr. Berryman Green, 537 Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month, 2:30 p. m., September to May, inclusive.

GEORGIA PEDIATRIC SOCIETY

President: Dr. Joseph Yampolsky, 127 Peachtree St., Atlanta.
Secretary: Nevin M. Atkins, Atlanta.
Place: Savannah, Ga. Time: May 9, 1928.

PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

President: Dr. William W. Anderson, 362 Peachtree St., Atlanta.
Secretary: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St., Atlanta.
Time: Second Thursday of each month from October to April, at 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. James Herbert Young, 66 Commonwealth Ave., Boston.
Secretary: Dr. Thomas Lanman, 286 Marlborough St., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Maurice Blatt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. J. K. Calvin, 104 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. L. R. De Buys, 1487 Delachaise St., New Orleans.
Secretary: Dr. F. J. Kinberger, 3601 Prytania St., New Orleans.

DETROIT PEDIATRIC SOCIETY

President: Dr. George E. White, Capitol Theatre Bldg., Windsor, Ontario, Canada.
Secretary: Dr. W. S. O'Donnell, 1563 David Whitney Bldg., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. F. W. Schlutz, University of Minnesota, Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester. Time: Winter, Spring, Summer, Fall.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary and Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, 731 City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, 1036 Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

BROOKLYN PEDIATRIC SOCIETY

President: Dr. Louis M. Ruderman, 961 Eastern Parkway, Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

BRONX PEDIATRIC SOCIETY

President: Dr. William Hinz, 1964 Grand Concourse, New York.
Secretary: L. H. Barenberg, 1749 Grand Concourse, New York.
Place: Concourse Plaza, 161st, St., and Grand Concourse, New York. Time: Second Wednesday of each month, except June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady, N. Y.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany, N. Y.
Place: Various Cities in New York. Time: Fourth Thursday of October, January and April.

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

President: Dr. William Ropes May, 8 East 66th St., New York.
Secretary: Dr. Stafford McLean, 17 East 71st St., New York.
Place: New York Academy of Medicine, 2 East 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

THE PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

Chairman: Dr. J. W. Epstein, 3912 Prospect Ave., Cleveland.
Secretary: Dr. Charles W. Burhans, 14805 Detroit Ave., Lakewood, O.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

PEDIATRIC SECTION, ACADEMY OF MEDICINE, TORONTO

President: Dr. M. A. Cox, 34 St. Clair Ave. West, Toronto.
Secretary: Dr. F. R. Hassard, 1361 King St. West, Toronto.
Time: November 17, December 15, February 16 and March 15.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Edward L. Bauer, Jefferson Medical College, Philadelphia.
Secretary: Dr. John Porter Scott, 2040 Pine St., Philadelphia.
Place: College of Physicians, 19 S. 22nd St., Philadelphia. Time: Second Tuesday of each month from October to June, inclusive.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Blue, 1024 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gilbert J. Levy, 899 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

TEXAS PEDIATRIC SOCIETY

President: Dr. Lucius D. Hill, Medical Arts Bldg., San Antonio.
Secretary and Treasurer: Dr. Edwin G. Schwarz, Medical Arts Bldg., Fort Worth.
Place: Galveston. Time: May 9, 1928.

RICHMOND PEDIATRIC SOCIETY

President: Dr. Howard Urbach, 1331 Grove Ave., Richmond, Va.
Secretary: Dr. W. Ambrose McGee, West End Medical Bldg., 2618 Grove Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St., Richmond, Va.
Time: First Thursday of each month (other than during the summer).

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Lawrence T. Royster, University, Va.
Secretary: Dr. William B. McIlwaine, Petersburg, Pa.
Place: Danville, Va. Time: October 17, 1928.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. R. S. Miles, Fidelity Bldg., Tacoma, Wash.
Secretary: Dr. D. M. Dayton, 704 St. Helens Ave., Tacoma, Wash.

SEATTLE PEDIATRIC SOCIETY

President: Dr. George B. McCulloch, Cobb Bldg., Seattle.
Secretary: Dr. C. L. Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, at 7 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. A. B. Schwartz, 418 E. North Ave., Milwaukee.
Secretary: Dr. M. G. Peterman, 167 Seventeenth St., Milwaukee.
Place: Children's Hospital, Milwaukee. Time: Second Wednesday of each alternate month, beginning with February.